Pine and Juniper Diseases in the Great Plains

Glenn W. Peterson

General Technical Report RM-86
Rocky Mountain Forest and
Range Experiment Station
Forest Service
U.S. Department of Agriculture

Abstract

The results of research on diseases of pines and junipers in the Great Plains are presented; diagnosis, biology, damage and control are emphasized.

Acknowledgments

Research on tree diseases in Lincoln has been greatly facilitated by Wayne Willmeng and other city foresters of Lincoln, Nebraska, who provided experimental tracts and field equipment.

The Horticulture and Forestry Department, University of Nebraska—Lincoln, has aided greatly by providing space for and maintaining experimental plantings.

Thanks are due Dr. Merton Brown, Jr., University of Missouri—Columbia, who provided photographs for figures (1-9, 1-12, 6-7, 7-12), and to Dr. William Merrill, Pennsylvania State University, for a photograph for figure 3-1.

Finally, the author acknowledges the excellent assistance of biological laboratory technician Michael Kuhl for his many contributions to tree disease research in this project for the past 14 years.

USDA Forest Service
General Technical Report RM-86

August 1981

Pine and Juniper Diseases in the Great Plains

Glenn W. Peterson, Plant Pathologist
Rocky Mountain Forest and Range Experiment Station[1]

[1]*Headquarters is in Fort Collins, in cooperation with Colorado State University; author is project leader of the Station's Research Unit at Lincoln, Nebr., in cooperation with the University of Nebraska—Lincoln.*

Contents **Page**

Pine and Juniper Diseases in the Great Plains

Glenn W. Peterson

Introduction

Pines and junipers are widely used in windbreak, shelterbelt, park, landscape, Christmas tree, roadside, wildlife, and other types of plantings in the Great Plains. The pines and junipers are especially valuable in plantings established for the protection of soil, crops, livestock, wildlife, and dwellings, because they provide protection all year.

Research on a number of diseases of pines and junipers throughout the Great Plains has been conducted since 1958 by the USDA Forest Service's tree disease project in Lincoln, Nebr., in cooperation with the Nebraska Agricultural Experiment Station. This publication summarizes the major results of the research.

Emphasis has been placed on results which pertain to diagnosing the diseases and controlling them. The first sections under each disease cover damage, geographic distribution, trees affected, and control. This information is included primarily for those who need to apply the information in the field. The last section under each disease is devoted to physiological and morphological information on the pathogens. Such information is included for researchers and other specialists who will be working with the pathogens in laboratories, greenhouses, and growth chambers.

1. Dothistroma Needle Blight of Pines

Pines affected by Dothistroma blight lose needles prematurely, thereby reducing growth. Repeated attacks sometimes kill trees. The infected needles become discolored (brown) prior to being cast. Some of the discolored needles remain on trees for months, thereby destroying the esthetic value of pines in landscapes and making Christmas trees unmarketable (fig. 1-1).

Dothistroma pini, the fungus causing this blight, affects several pine species in 21 states in the United States, including southeastern Alaska (fig. 1-2). Ponderosa and Austrian pines, which are highly susceptible, have been severely damaged in the Great Plains. Scots pine is highly resistant to *D. pini*. This fungus is present in some natural pine stands, but it causes more damage to planted pines. In the Great Plains, the disease is encountered in windbreaks, and Christmas tree, roadside, park and other plantings.

Diagnosis

Symptoms of Dothistroma blight in the central United States develop in the fall of the year of infection. Usually, infection is more intense in the lower crown of trees (fig. 1-3), but the entire crown can be infected (fig. 1-1), even in 35-year-old trees.

Early symptoms on needles consist of deep green bands (fig. 1-4) and yellow and tan spots. The spots and bands may turn brown to reddish-brown (fig. 1-5). The reddish bands are more distinctive and numerous on infected pines in the western United States (California, Oregon, Washington, Idaho) where this disease is often referred to as the "red band" disease.

Commonly, the ends of infected needles turn light green, then yellow, and then necrotic (brown), with the base of the needles remaining green (fig. 1-6). Needles may develop extensive necrosis (browning) 2 to 3 weeks after the symptoms first appear.

Spores of the fungus are borne in fruiting bodies, which develop below the epidermis of needles. The fruiting bodies develop in lesions (fig. 1-7). As the fruiting bodies enlarge, they split the epidermis longitudinally along two sides of the fruiting body (figs. 1-8, 1-9). The earliest developing fruiting bodies are frequently located near the margin between brown and green tissues (fig. 1-10). In the central United States, these fruiting bodies may enlarge sufficiently in late fall to split the epidermis. They generally do not mature and produce spores until the following spring, however.

Infected needles drop prematurely. Infected second-year needles are cast before infected first-year (current-year) needles. In some years, second-year needles are cast in late fall of the year they become infected. In other years, loss of needles is not extensive until late the following spring or early summer. Needles that become infected the year they emerge often are not shed until late summer the following year.

In the central United States, trees may become infected from May to October. Although infection can occur early in the growing season, symptoms do not develop before September. Symptom development is usually not extensive until November or December.

Damage caused by *D. pini* may be confused with damage from other causes. Pines under moisture stress frequently have needles with brown tips. The brown tips of such needles are usually severely constricted, whereas tips of Dothistroma-infected needles are only slightly constricted. The demarcation between discolored tips and green tissue is sharp in Dothistroma-infected needles, but is gradual in needles under moisture stress.

Needles damaged mechanically, such as by spray or cultivation equipment, may have bands, spots, and brown tips. Similar damage can be caused by some insects. Dothistroma may be distinguished from such damage by the presence of fruiting bodies. These dark-colored bodies can be seen in the spots and bands.

It may be difficult to distinguish Dothistroma blight from brown-spot needle blight caused by the fungus *Scirrhia acicola*. The symptoms of these two diseases are similar. Both cause spots, bands, and dead needle

tips. Symptoms of brown spot develop earlier than symptoms of Dothistroma. Symptoms of brown spot were observed in July, in eastern Nebraska, in 1971, when Dothistroma symptoms were not evident before October. These two fungi are best distinguished by differences in their spores. Both fungi produce long cylindrical conidia (spores) which have from 1 to 5 cross walls (septations); however, *D. pini* conidia are transparent (hyaline), whereas *S. acicola* conidia are greenish-brown. There are also differences in size of conidia of the two fungi. A specialist may have to be consulted to make the distinction.

Life Cycle

Spores of *D. pini* are dispersed from May to October, in the central United States (figs. 1-11, 1-12). The spores are dispersed primarily by rain splash and, therefore, are not dispersed long distances. Spores deposited on needles germinate when temperature and moisture conditions are favorable. The spores produce germ tubes which grow in a positive direction toward stomates through which they enter needles (figs. 1-13, 1-14, 1-15).

Second-year and older needles of ponderosa and Austrian pines are susceptible from late May through the growing season in the central United States. However, first-year (current-year) needles of these two pines are not susceptible until mid-July. Fruiting bodies of the fungus develop after symptoms appear. In the central United States, spores are rarely formed in the fall of the year of infection. Spores which are formed in the fall do not cause infection in the fall, because conditions at that time are not favorable for infection. In the following spring, fruiting bodies mature and spores develop within them. These spores then initiate new infections, and the disease cycle is repeated.

Dothistroma pini is spread to new areas, in most cases, by infected nursery stock. There is considerable evidence that the fungus is spread by distribution of 1- to 3-year-old seedlings. Infection in stock this age is often low and difficult to detect. A survey of 60 Christmas tree plantings in Nebraska revealed that half of the plantings contained Dothistroma-infected trees. Because these plantings were located far from other pines, planting stock appears to be the source of infection. Infection in some planting stock was so low that growers were not aware of blight in their plantations until 4 or 5 years after planting. It is unlikely that long distance spread is caused by spores being carried by wind from infected trees to new plantings. In eastern Nebraska no infection has occurred in a ponderosa pine planting located 1 km from an Austrian pine plantation that has been severely infected since 1967.

Control

Dothistroma pini can be controlled by proper timing of fungicide treatments, just before the period of susceptibility (fig. 1-16). Because second-year and older needles of Austrian and ponderosa pines are susceptible in late May, fungicide applied in mid-May will protect these older needles. Fungicide applied in mid-May will not protect new needles, however, because at that time they are very small (just emerging from fascicle sheaths). New needles of Austrian and ponderosa pines do not have to be covered with fungicides early in the season because they are not susceptible until mid-July (fig. 1-16). A second application of fungicide in mid-June will effectively protect new needles.

There is a low risk in using just one fungicide application. Results from research in eastern Nebraska show that one application in mid-June will provide satisfactory control in most years, although older needles will be unprotected for a short period (late May to mid-June). In eastern Nebraska, initial infection did not occur until about mid-June in four out of five years. Many Christmas tree growers in the central United States are effectively controlling Dothistroma with a single fungicide application.

Annual spraying for control of *Dothistroma pini* is unnecessary in certain types of plantings. Because essentially complete control of this pathogen can be obtained with fungicides, managers can risk not spraying every year in park, residential, and similar types of plantings. If infection occurs during the year in which fungicide is not applied, fungicide can be applied the next year, knowing good control will be obtained. If no or little infection occurs the year skipped, then another year can be skipped. However, Christmas tree growers probably should not skip any years because of the possibility of high financial loss.

Copper fungicides prevent infection by *D. pini*. Bordeaux mixture (8 pounds of copper sulfate, 8 pounds of hydrated lime, and 100 gallons of water) is very effective and is registered for control of Dothistroma. Fungicides containing copper salts of fatty and rosin acids are also registered for Dothistroma control.

The impact of *D. pini* can be reduced by use of resistant trees. Pine species differ in resistance to *D. pini*. Scots and red pines are highly resistant, whereas ponderosa and Austrian pines are highly susceptible (fig. 1-17).

Resistance to the fungus varies considerably within species. An evaluation of 22 geographic sources of *Pinus nigra* revealed that some trees from 16 of the sources had high resistance; all trees of one geographic source had high resistance (fig 1-18). Seedlings are now being produced in the Great Plains from seed collected in an area in Yugoslavia where the resistant trees originated. Resistance to Dothistroma is being evaluated in 50 geographic sources of ponderosa pine in eastern Nebraska.

The pattern of resistance varies considerably in Austrian and ponderosa pines. On some trees, needles of all ages are highly resistant. On other trees, current-year needles are resistant, but older needles are susceptible (fig. 1-19).

Physiology and Morphology

The fungus has both a sexual stage (*Scirrhia pini*) and an asexual stage (*Dothistroma pini*). The sexual

stage has been found in British Columbia, Alaska, Oregon, and California but not elsewhere in North America. Ascospores are produced by the sexual stage; their role in development of epidemics is not known. Conidia (spores produced by the asexual stage) are cylindrical, curved, 1- to 5-septate but usually 3-septate, and hyaline (fig. 1-11). The conidia from the central United States are considerably shorter on the average than those found in the western United States. The average dimensions of conidia in seven separate collections from Nebraska were 21.3 μm $\times$ 2.5 μm.

Conidia from Nebraska collections germinated over the temperature range 12-28°C when incubated on water agar. Conidia incubated on water agar had optimum germination at 24°C (fig. 1-20). Germination was near optimum at 22°C but dropped off rapidly below 22°C and above 24°C. Germ tube growth on water agar was optimum at 22-24°C (fig. 1-20).

Conidia incubated in water had optimum germination at 22-24°C; germ tube growth was optimum at 24°C. Germ tubes growing in water at 22°C and 26°C were markedly shorter than those at 24°C.

Conidia incubated on water agar germinated earlier than conidia incubated in water; germ tubes grew faster on water agar. At 24°C conidia on water agar started to germinate between 6 and 8 hours after incubation started (fig. 1-21), whereas conidia in water started to germinate between 8 and 10 hours after incubation started. These results show that when comparing germination of *D. pini* conidia from different collections, results can be obtained quicker if spores are germinated on water agar rather than in water.

The optimum temperature for germination of *D. pini* conidia has been reported to be near 18°C in New Zealand and in Kenya. The germination tests in these countries involved incubation in water, whereas, in the Great Plains, conidia were incubated on water agar. To see if methods might account for the differences, 10 isolates were incubated on both water agar and in water. Because the optima were similar in water and on water agar, the difference in optima are likely real and not a result of differences in germination test methods. Accordingly, there is a possibility that the lower temperature (18°C) for optimum germination of conidia in New Zealand and Kenya results in more infection at lower temperatures in those countries than in the central United States, where the optimum for germination is 24°C.

Growth of the fungus on malt extract agar was best at 20°C; growth was moderately good at 16°, 22°, and 24°C.

Figure 1-1.—Austrian pines infected by *Dothistroma pini*.

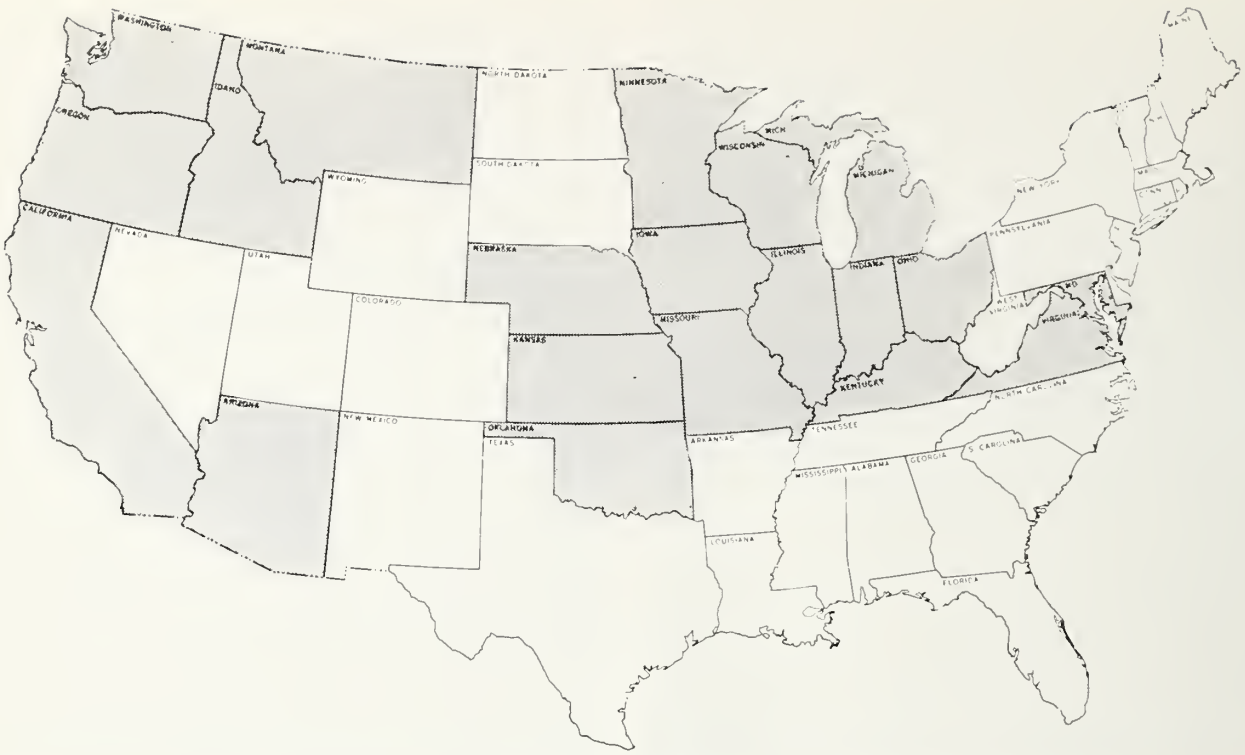

Figure 1-2.—*Dothistroma pini* occurs in shaded states and in southeastern Alaska.

Figure 1-3.—Infection of the lower crowns of ponderosa pines in a field windbreak.

Figure 1-4.—Early symptoms of Dothistroma blight—deep green bands.

Figure 1-5.—Typical symptoms of Dothistroma blight—reddish brown spots and bands.

Figure 1-6.—Symptoms typical of *Dothistroma pini* infected needles—tips brown, bases green.

Figure 1-7.—Lesion on infected needle with an erumpent fruiting body of *Dothistroma pini*.

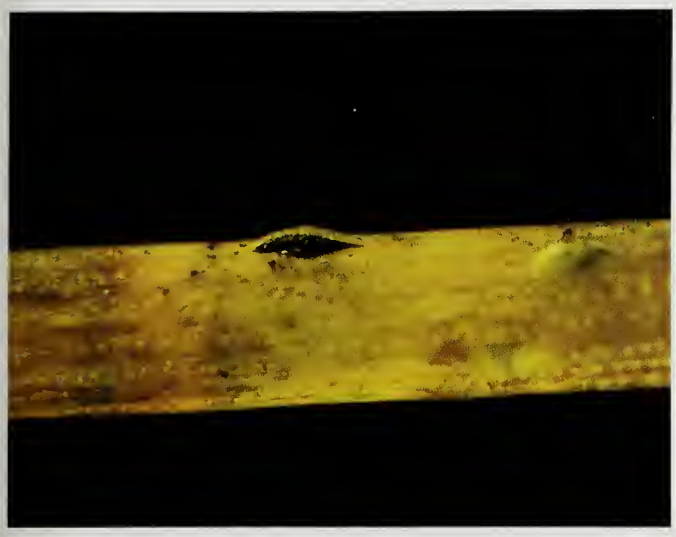

Figure 1-8.—Needle epidermis split and raised by developing fruiting body of *Dothistroma pini*.

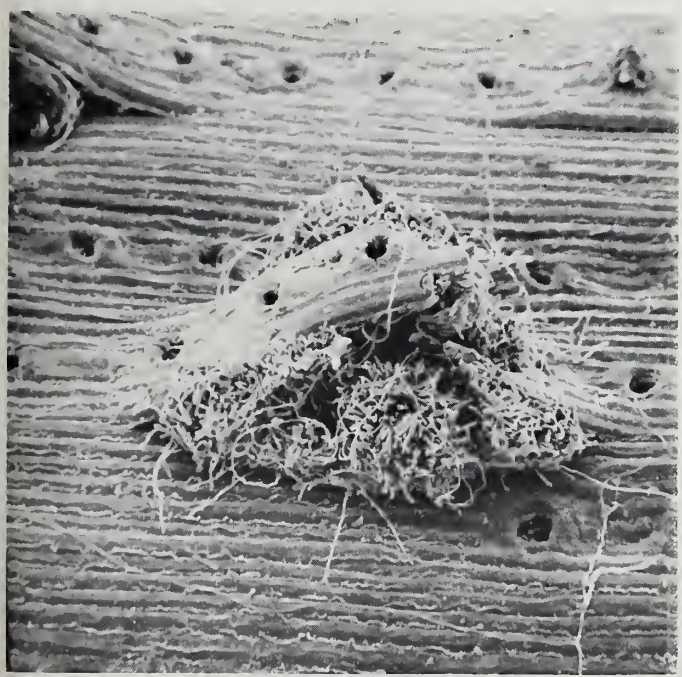

Figure 1-9.—Flap of split epidermis raised by *Dothistroma pini* fruiting body. (X200)

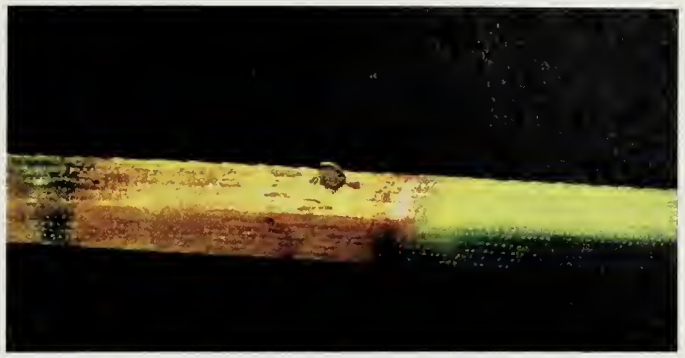

Figure 1-10.—Erumpent fruiting body of *Dothistroma pini* located near margin between brown and green tissues.

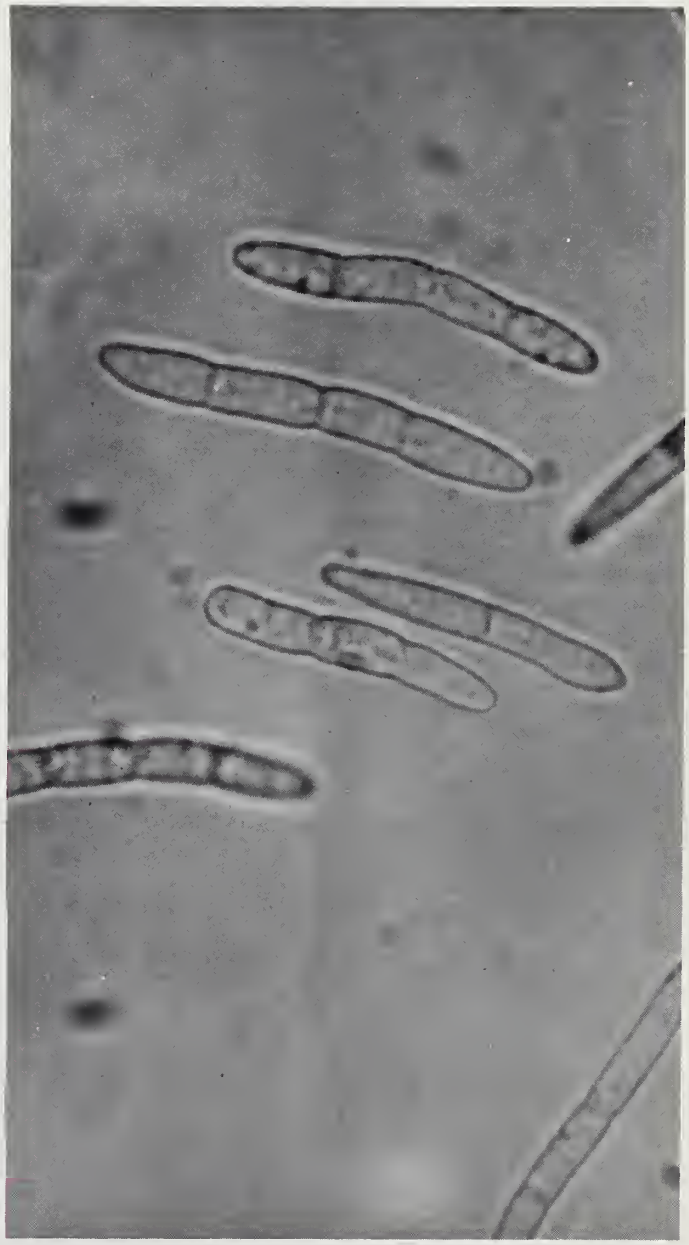

Figure 1-11.—Conidia of *Dothistroma pini*, stained with cotton blue to show cross walls.

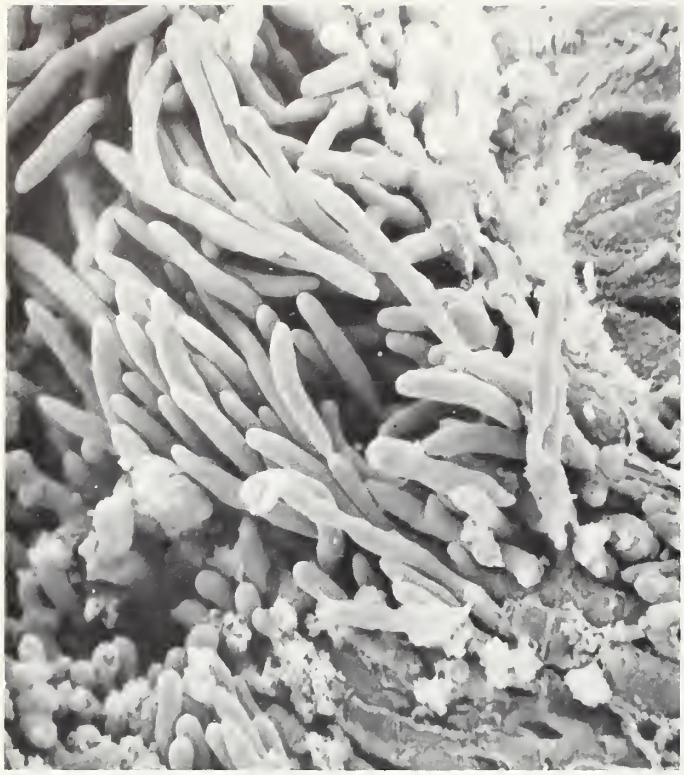

Figure 1-12.—Spores (conidia) of *Dothistroma pini.* (X2000)

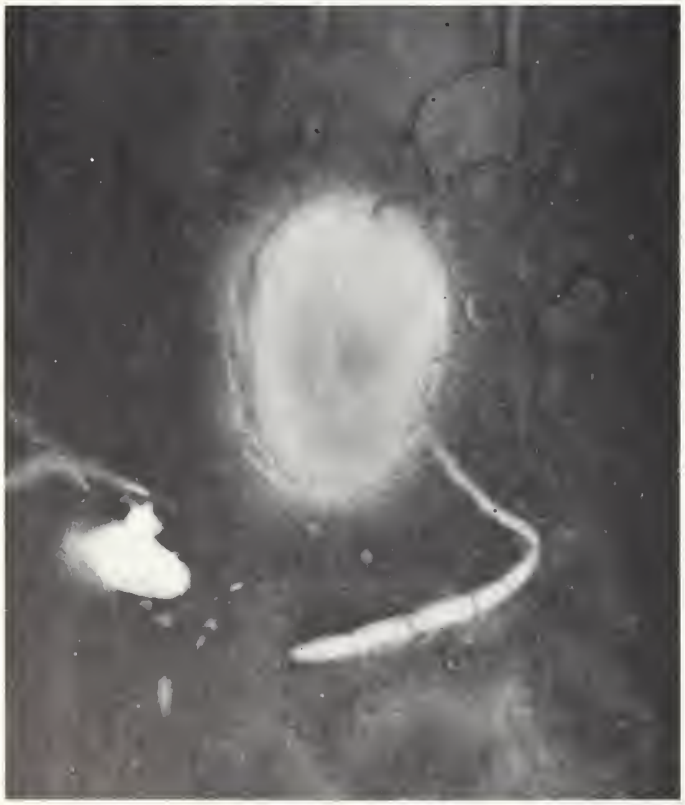

Figure 1-13.—Germinated conidium of *Dothistroma pini* with germ tube growing toward a stomate, on an Austrian pine needle.

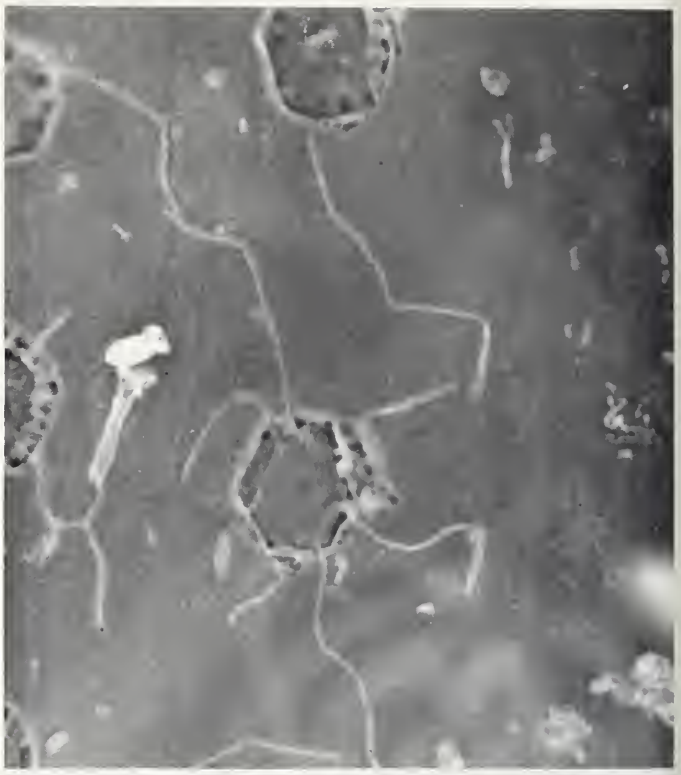

Figure 1-14.—Germ tubes from several conidia of *Dothistroma pini* growing in a positive direction toward stomates, on an Austrian pine needle collected in the field.

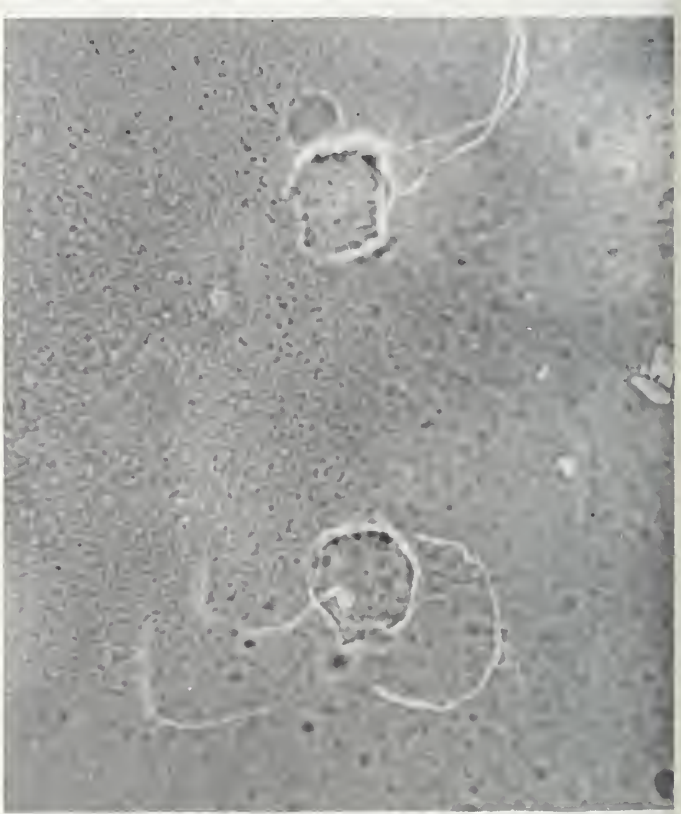

Figure 1-15.—Germ tubes of *Dothistroma pini* curving towards stomates on an Austrian pine needle.

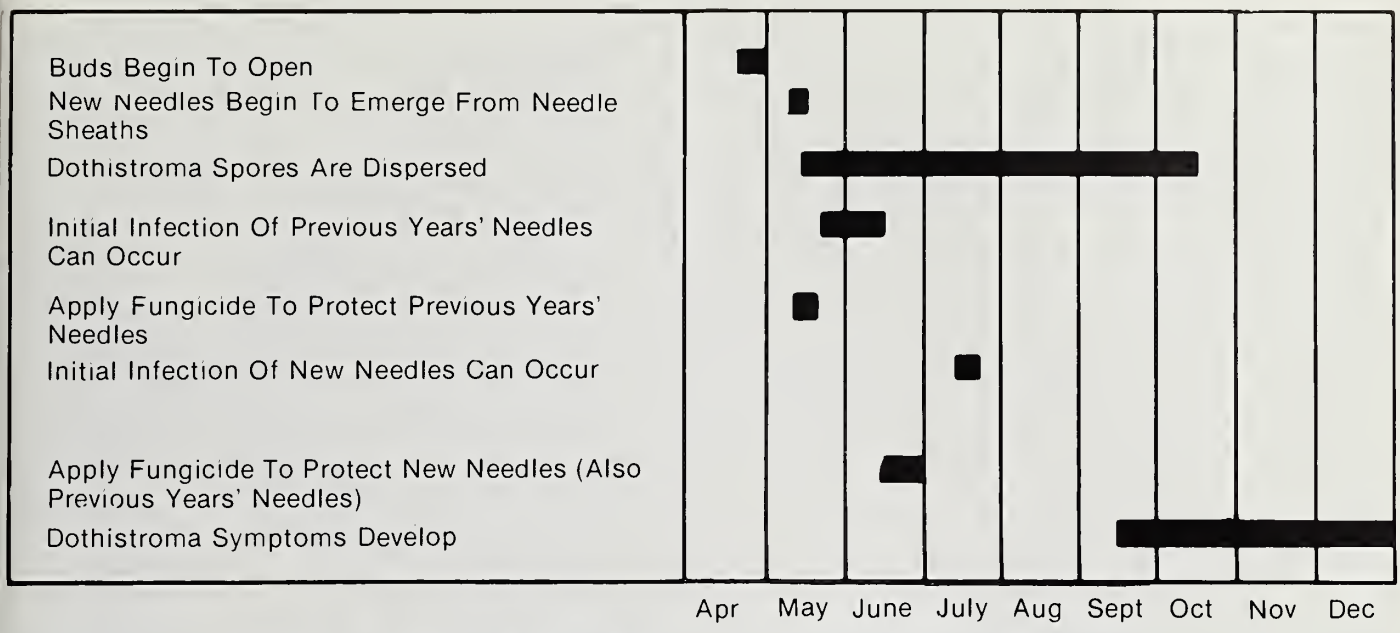

Figure 1-16.—Schedule for developing programs for control of Dothistroma blight.

Figure 1-17.—Resistant Scots pines (left) adjacent to susceptible Austrian pines (right).

Figure 1-18.—A Yugoslavian source of *Pinus nigra* resistant to *Dothistroma pini* (background) adjacent to a suspectible Spanish source (foreground).

Figure 1-19.—Austrian pine with resistant first-year (current-year) needles and susceptible second-year needles (Photographed in November).

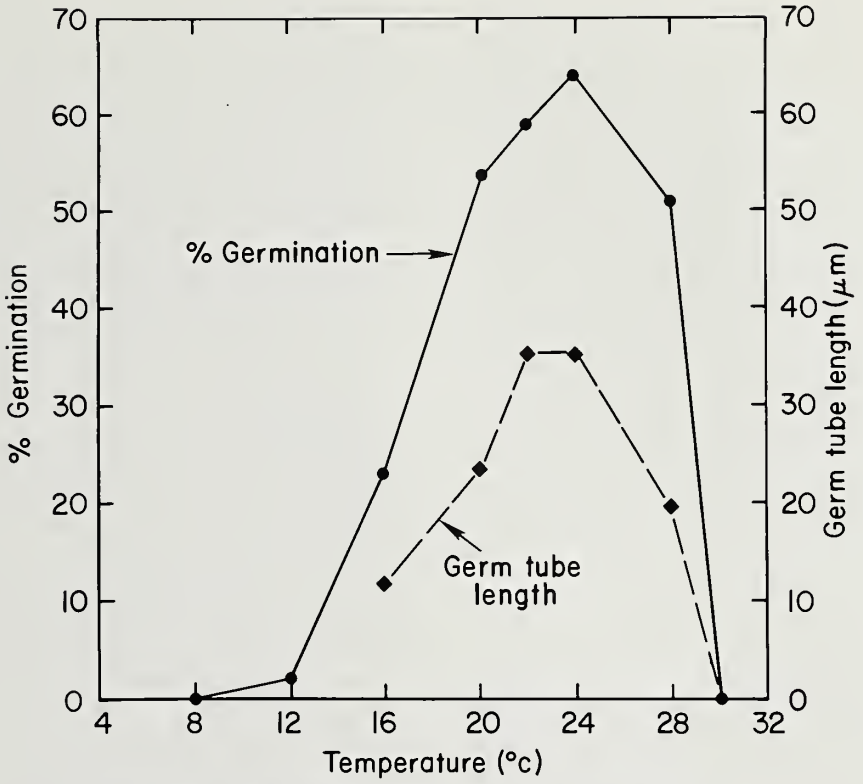

Figure 1·20.—Effect of temperature on germination of *Dothistroma pini* conidia incubated for 24 hours on water agar.

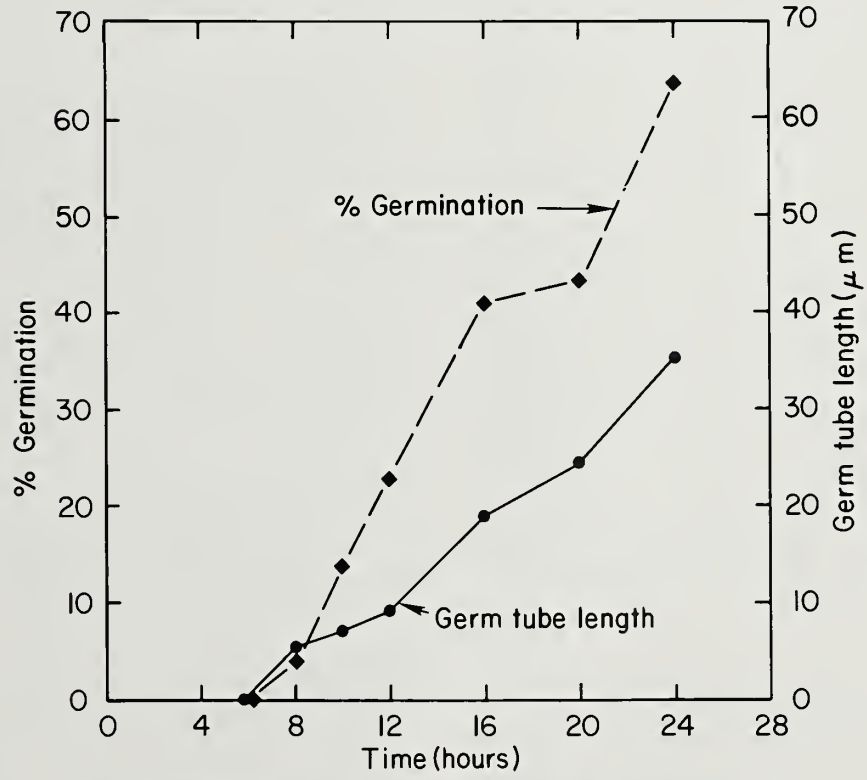

Figure 1·21.—Germ tube length and percent germination of *Dothistroma pini* conidia, incubated for various periods of time, on water agar, at 24°C.

2. Brown Spot Needle Blight of Pines

Brown spot disease caused by the fungus *Scirrhia acicola* has affected pines in the Great Plains, particularly pines in Christmas tree and landscape plantings. Ponderosa and Scots pines have been the hosts most commonly infected in the Great Plains.

The disease caused by this fungus is very similar to the blight caused by *Dothistroma pini*. Infected needles are cast prematurely, and prior to being cast, become discolored (brown). Some of the discolored needles remain on trees for months, thereby destroying the esthetic value of pines in landscapes and making Christmas trees unmarketable.

The most severe damage by the brown spot fungus occurs outside the Great Plains. Large economic losses caused by this fungus occur in plantings of longleaf pine in southern states. Damage to Scots pine in Christmas tree plantings has been more severe in states east of the Great Plains.

Diagnosis

Brownish spots and bands develop in infected Scots and ponderosa pine needles. The tips of the needles become tan, then necrotic, with the bases of the needles remaining green. Later, the bases turn brown. Most diseased needles are cast in the fall, but some are not cast until the following growing season.

Symptoms and damage caused by *S. acicola* are similar to those caused by *D. pini*. *Dothistroma pini* infected needles also develop spots and bands, and the tips of needles become necrotic first, with bases remaining green. Symptoms of brown spot develop earlier than do symptoms of Dothistroma blight. Initial symptoms developed in July, on Scots pine, in a provenance planting, in eastern Nebraska, during the same year, and on the same experimental tract, in which initial symptoms of Dothistroma blight on Austrian pine did not develop until October.

Scots pine, which is highly susceptible to *S. acicola*, is highly resistant to *D. pini*. Ponderosa pine, however, is highly susceptible to both fungi. It is very difficult to distinguish the two blights on ponderosa. Not only are the symptoms similar, but the fruiting bodies are also similar. These two fungi are best distinguished by differences in their spores. Both fungi produce long, cylindrical conidia (spores) which have from 1 to 5 (usually 3) cross walls; however, *S. acicola* conidia are greenish brown, whereas *D. pini* conidia are transparent (hyaline). There are also differences in size of the conidia of the two fungi. A specialist may have to be consulted to make the distinction.

Life Cycle

The primary inoculum is spores which develop in the spring, from fruiting bodies formed after infection the previous growing season. The spores are dispersed principally by rain splash, and are not dispersed long distances. Investigators working in Wisconsin found that generally there are two periods when many spores are dispersed — one in June and one in late August-September (Skilling and Nicholls 1974). They also found that young, growing, Scots pine needles are more susceptible to infection than are mature needles. Therefore, spores released late in the summer apparently cause little infection, because the current-year needles are more resistant than they were earlier in the year. Severe infection occurred between June 26 and July 24, but infection was slight after July 24.

The short-needled varieties of Scots pine, particularly those from France and Spain, are especially susceptible to *S. acicola*. In a planting of 36 geographic sources of Scots pine the short-needled Spanish and French sources were the first to have a significant level of infection.

Scots pine from these geographic sources are growing on an experimental tract which also contains 22 geographic sources of *P. nigra*. The level of infection by *S. acicola*, even in Scots pines from the most susceptible geographic sources, has been much lower than the level of infection by *D. pini* in 21 of the 22 geographic sources of Austrian pine.

However, infection of Scots pines by *S. acicola*, in some Christmas tree plantings, in southeastern Nebraska and in northeastern Kansas, has reached the level requiring control by fungicides. Ponderosa pine also has been heavily infected in southwestern Missouri.

Control

Protective fungicides will control the brown spot needle blight fungus. Several fungicides are effective including Bordeaux mixture and chlorothalonil (Bravo, Daconil),[2] which are EPA registered. In the north-central region, a single application in mid-June is usually sufficient. In severely infected plantations or during unusually wet years, a second spray applied 3 to 4 weeks later may be required.

Where brown spot disease is a serious threat, highly susceptible varieties of Scots pine, such as some of the short-needled Spanish and French varieties, should not be planted.

Physiology and Morphology

Readers are referred to the bulletin by Siggers (1944) and the article by Wolf and Barbour (1941) for information on the physiology and morphology of *S. acicola*.

[2]*Mention of a trade name or proprietary product does not constitute a guarantee or warranty by the U.S. Department of Agriculture, and does not imply its approval to the exclusion of other products that also may be suitable.*

3. Naemacyclus Needle Cast of Pines

The fungus *Naemacyclus minor* occasionally has caused premature needle cast of pines in the Great Plains. Moderate damage has been found in pines, in Christmas tree plantings, in Kansas, Nebraska, and South Dakota. Hosts in these plantings include Austrian, ponderosa, and Scots pines. This fungus has often been considered a weak parasite; yet, it is currently considered the most damaging pathogen in Scots pine Christmas tree plantings in Pennsylvania. Considerable research has been conducted on the disease by plant pathologists at Pennsylvania State University.

Diagnosis

Diagnosis is relatively easy because of the distinctive fruiting bodies (apothecia) which form on necrotic needles. The fruiting bodies arch upward in a cushion shape, so that the epidermis lying above them splits longitudinally along lines of stomates. The torn epidermal pieces are flapped outwards (fig. 3-1). The torn epidermis pieces can be observed with the unaided eye and can readily be seen with a hand lens. The upper surface of the fruiting body is dull, cream white.

Life Cycle

Investigations of the life cycle of this fungus have been conducted in Pennsylvania but not in the Great Plains. The information on the life cycle presented here has been drawn from the research in Pennsylvania (Kistler and Merrill 1978).

Symptoms develop in September and October after a 10- to 15-month incubation period. The first symptoms are small, light-green spots which develop in September. The spots enlarge and lighten in color; eventually the whole needle becomes yellow with prominent, transverse, brown bands. Symptoms develop first on the second-year needles and on third-year needles not infected the previous year. Foliage of severely infected pines appears distinctly yellow. Most of the infected second-year and third-year needles are cast in October and November, and the characteristic waxy fruiting bodies then form on the cast needles. Some infected necrotic needles remain attached to the tree throughout the winter. These attached needles may bear fruiting bodies, or fruiting bodies may develop on them the following spring. Severely diseased trees retain only the current year's needles.

Ascospores of *N. minor* are released throughout the growing season. The work in Pennsylvania indicates that the principal infection period is July-August. Isolations from needles of different ages have shown that *N. minor* is present in a high percentage of symptomless needles. On many trees, there is no external evidence that the needles have been infected until fruiting bodies develop on them, after they have been normally (not prematurely) cast. In Nebraska, fruiting bodies of *N. minor* have been found on normally cast needles of healthy ponderosa and Scots pines.

An experimental planting in North Dakota, containing many geographic sources of Scots pines, was severely diseased by *N. minor* (fig. 3-2) in the early 1970's. An experiment was established in 1974 to obtain information on when infection occurs and how the disease could be controlled. There had been extensive premature needle casting in the planting in 1973. Most of the second-year needles were necrotic (fig. 3-3). However no new damage occurred in the planting in 1974, nor has there been any significant damage by *N. minor* in the planting since 1974. The reason for the absence of disease in the planting is not known; however, there are several possible explanations. Every other tree (50% of all trees) in the planting was removed in the summer of 1973. The change in moisture conditions resulting from tree removal might have been unfavorable for infection. Also, reduction in moisture stress on trees in the 10-year-old planting might have been involved. Perhaps the weather conditions (rain, temperature) since the epidemic have not been favorable for infection.

Control

The Naemacyclus needle cast fungus can be controlled by protective fungicides. Investigators in Pennsylvania have found two fungicides that are effective. Registration of these fungicides will be sought when their research on timing of fungicide application is completed, probably in 1982. The timing of the first application depends upon conditions which influence the development of fruiting bodies and spores of the fungus. In some years conditions favorable for development occur in early April in Pennsylvania; thus the initial fungicide application should be made by that time. Depending on the year and the fungicide used, control has been obtained with from one to three fungicide applications.

Physiology and Morphology

After an extensive morphological and taxonomical study of *Naemacyclus niveus* and related species, H. Butin suggested that two species of *Naemacyclus* exist on pine needles. The one species, *N. niveus*, was described in the last century; the other species, *N. minor*, was described by Butin in 1973. The *Naemacyclus* species found on ponderosa and Scots pines in the Great Plains is considered to be *N. minor*. The mycelial growth of Great Plains isolates is as described by Butin for *N. minor*, that is thick aerial mycelium develops which later collapses so that the hyphal layer appears dissolved. The centers of colonies are dark brown. Apothecia form in culture.

The following is from Butin's description: asci are cylindrical to club-shaped, 110-115 μm long, 11 μm wide with 8 ascospores; ascospores are hyaline, often twisted, 65-98 μm long, 2.5-3.0 wide, with two septa, paraphyses are filiform, branched at the tip and light yellow. The average length of the 30 ascospores from each of 12 collections in the Great Plains ranged from 70 μm to 89 μm. The length of ascospores of all isolates averaged 80 μm. These data are similar to those reported by Butin.

Pennsylvania investigators report the optimum temperature for mycelial growth *in vitro* as 25°C and for ascospore germination as 22°C.

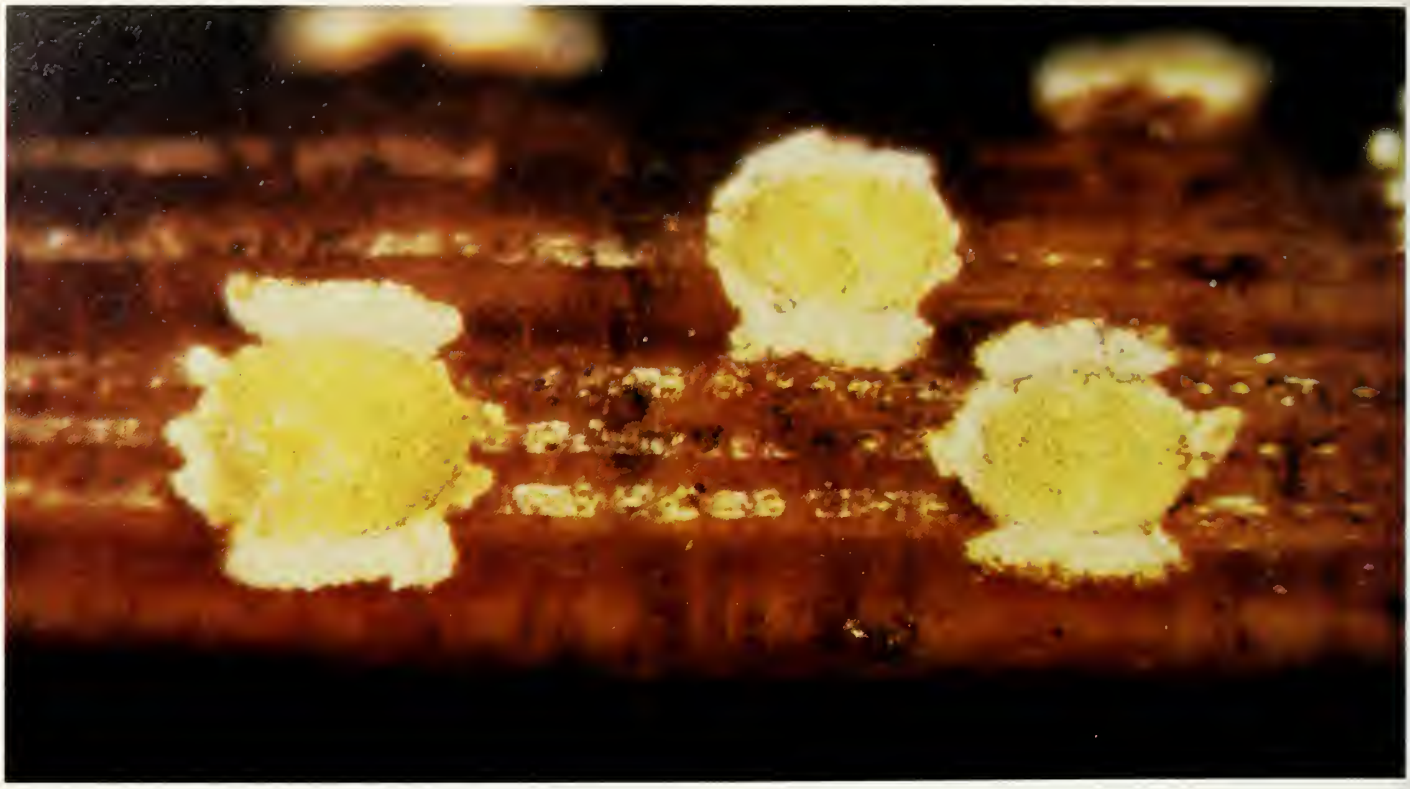

Figure 3-1.—Fruiting bodies of *Naemacyclus minor* on Scots pine needle.

Figure 3-2.—Scots pine infected by the needle cast fungus *Naemacyclus minor.*

Figure 3-3.—Previous years' needles of Scots pine infected by *Naemacyclus minor.*

4. *Diplodia pinea* Blight of Pines

The fungus causing Diplodia blight infects new shoots of pines. Commonly, the entire new shoot is killed. Older stem tissues also become infected, with the result that major branches are killed back to the main stem. This fungus can destroy the protective value of pines in shelterbelts and windbreaks and the esthetic value of pines in landscapes (fig. 4-1).

Diplodia pinea is widely distributed in the United States (fig. 4-2). Many pine species are infected by this fungus; however, major damage has occurred in plantings of exotics. In the United States, it has been most frequently reported on Austrian pine, which has been widely used in landscape plantings. Other common hosts in the United States are ponderosa, Scots, red, Monterey, and mugo pines. Damage does occur to native species but primarily in plantings; this fungus has seldom been reported in natural pine stands.

Diagnosis

Diagnosis of *Diplodia pinea* blight is relatively easy. Symptoms of the disease are distinctive, and fruiting body location and appearance are such that confusion with other fungi is unlikely.

Droplets of resin usually can be found on infected new shoots before the needles have broken through fascicle sheaths (fig. 4-3). A close examination of such shoots usually reveals that one or a few needles are shorter and darker in color than the rest.

The fungus rapidly invades and kills all needles and tissues of new shoots (fig. 4-4). Commonly, this will occur after the needles have broken through fascicle sheaths but well before needles have reached full size. Early June is a good time to check for new infections.

Because new shoots can be killed by other agents, additional information is needed to determine whether the observed damage is by *D. pinea*. The best field evidence comes from observation of fruiting bodies of the fungus. The dark fruiting bodies (pycnidia) erupt through the epidermis. They usually are numerous at the base of needles and are particularly numerous on the section of the needle covered by the fascicle sheath (fig. 4-5).

The time of appearance of erumpent fruiting bodies on new shoots varies considerably. Fruiting bodies are rarely found on newly infected needles before late summer or early fall. In some years, they are not numerous until the following spring. When symptoms observed in late spring or early summer on trees infected for the first time indicate *D. pinea* infection, pycnidia will not be present on needles. However, this problem can be overcome by placing needles with symptoms in a moist chamber for 48 to 72 hours. If the needles are infected with Diplodia, pycnidia will develop on the needles.

Pycnidial development is not uniform on all needles of a shoot killed by Diplodia. Needles which are ashen gray and which can be detached easily from the shoot should be checked for presence of pycnidia. Needles which are tan or reddish-brown are not suspect.

Whether Diplodia has infected the tree also can be determined by examining second-year or older seed cones. Pycnidia develop profusely on scales of these cones (fig. 4-6). However, pines may have infected second-year seed cones but not infected shoots.

For the specialist with access to microscopes and facilities for culturing fungi, additional identification checks can be made. First, a check can be made to see if the spores are typical of *D. pinea* in size and color (fig. 4-7). Spores of *D. pinea* are colorless (hyaline) when formed but become dark brown. There is a problem when considering another characteristic typical of spores of fungi in the genus *Diplodia*. Spores in the genus *Diplodia* usually are one-septate (two-celled); however, mature dark colored spores of *D. pinea* seldom are one-septate. Spores usually do not have any septations. They do have the potential for forming a septum, however.

Life Cycle

Spores are dispersed beginning before buds start to open and new shoots develop. Thus the fungus is ready to infect the new shoots as soon as they develop. Although shoots can be infected through wounds, infection of new shoots does not depend on wounding.

Diplodia pinea infects not only the tissues (foliage and stem) of new shoots, but infects seed cones as well. The seed cones of ponderosa, Austrian, and Scots pines are not susceptible the year they emerge but are susceptible the second year. Numerous fruiting bodies develop on scales of infected, second-year cones. The pycnidia on cones are an abundant source of inoculum for infection of new shoots. Damage to older pines is attributed to the fungus first infecting second-year seed cones, then spores from pycnidia on the seed cones infecting new shoots. Several years' observations have revealed that second-year seed cones may be infected before new shoots are significantly infected.

In the central Great Plains, damage usually does not become severe until after pines are 30 years old. Pines in plantings less than 30 years old have been damaged, but usually when younger plantings were established near older pines.

Infected seedlings in nurseries usually are in beds which have been established near older, cone-bearing pines. Container-grown pine seedlings placed in the shade of Diplodia-infected pines to harden-off have been destroyed by *D. pinea*.

Several years after shoots are first infected, *D. pinea* damage occurs to older stem tissues. This damage may extend the length of major branches, killing them back to the main stem (fig. 4-8). Occasionally there is extensive killing of branches at the top of trees (fig. 4-9). Commonly, all tissues of infected first-year shoots are killed back to the node. Although it reasonably might be expected that the fungus grows from the infected new shoots into adjacent and older internodes, periodic observations of branches containing infected new shoots did not reveal a single case in which the adjacent older tissues became infected. Histological studies of stem

tissues at the node between current-year growth and second-year growth, revealed that the fungus present in current-year growth did not grow into the second-year growth. *Diplodia pinea* can be readily isolated from older, damaged tissues. Wounds, such as those caused by hail, can become infected; thus wounds would make the older tissues vulnerable to infection. However, examination of infected older tissues has not revealed any obvious wounds, such as those caused by hail. Possibly there are obscure wounds, such as those made by some insects, which could be the infection courts.

Because seed cones become infected the second year, possibly the fungus grows into the second-year and older stem tissues via the peduncle of the cones. The fungus has been consistently isolated from the scales, axis, and peduncle of cones; however, the fungus has not been isolated from stem tissues subtending infected peduncles of cones.

When infected new shoots die, buds are frequently forced just beneath the dead shoot. A check of shoots developing from the forced buds has not revealed that they are an avenue by which the fungus enters older stem tissues.

Thus the avenue of infection of older stem tissues which are free of gross wounds is not known. Specific-aged internodes were inoculated to determine if the fungus will grow across proximal and distal nodes and damage adjacent internodes. The fungus did not grow into adjacent internodes.

Control

Infection of new shoots can be reduced effectively by properly timed applications of fungicide. The following information on infection and disease development has been useful in determining when fungicides should be sprayed (fig. 4-10).

Generally, the temperatures prevailing during the early period of shoot formation are favorable for spore germination and germ tube growth. Through field inoculations, made at different times, of newly developing shoots of Austrian, ponderosa, and Scots pines, infection was found to occur as soon as buds begin to open. Infection continued to occur until approximately mid-June. A series of tests extending over 3 years was made to determine when, during the period from bud opening (approximately April 24 in eastern Nebraska) to mid-June, the shoots were most susceptible. The period of susceptibility was found to be a 2-week period beginning with bud opening (fig. 4-10). This period in eastern Nebraska is approximately April 24 to May 8. Applying fungicide after May 15 was of no value.

Because cones are an abundant source of inoculum, removal of infected branches (sanitation) does not have a significant effect on infection intensity. Spores are produced on leaf and stem tissues, but the numbers produced are small in relation to the numbers produced on seed cones.

Fungicide applied to control shoot infection has not been effective in preventing infection of second-year cones. This is not surprising, because the seed cones still are rapidly expanding after the period (April 24 - May 8) in which fungicide is applied for control of shoot infection. Thus the surface of expanding cones is not adequately covered by the early fungicide applications.

Physiology and Morphology

Diplodia pinea grows rapidly on potato dextrose agar and other commonly used media. Colony growth was optimum at 28°C (fig. 4-11). A high percentage of conidia of *D. pinea* germinated in the temperature range 16°C to 36°C (fig. 4-12). Germ tube growth was greatest at 28°C. A high percentage of conidia germinated within 2 hours, and germ tube growth was very rapid after 2 hours (fig. 4-13).

Pycnidia develop when *D. pinea* is cultured on media such as potato dextrose agar; however cultures need to be incubated in the light. After 10 days, incubation at 28°C, under 15-watt fluorescent light, cultures contained 30 pycnidia per cm^2; viable spores were present in one-third of these pycnidia after 10 days.

Isolates vary in their capacity to produce pycnidia. Pycnidial production is often enhanced by growing the fungus on culture media to which sterile pine needles have been added.

The dimensions of D. pinea conidia are in the range 35-40 μm by 16-18 μm.

Figure 4-1.—Austrian pine tree infected with *Diplodia pinea*.

Figure 4-3.—Stunted needle with resin droplet resulting from initial infection of a new shoot by *Diplodia pinea*.

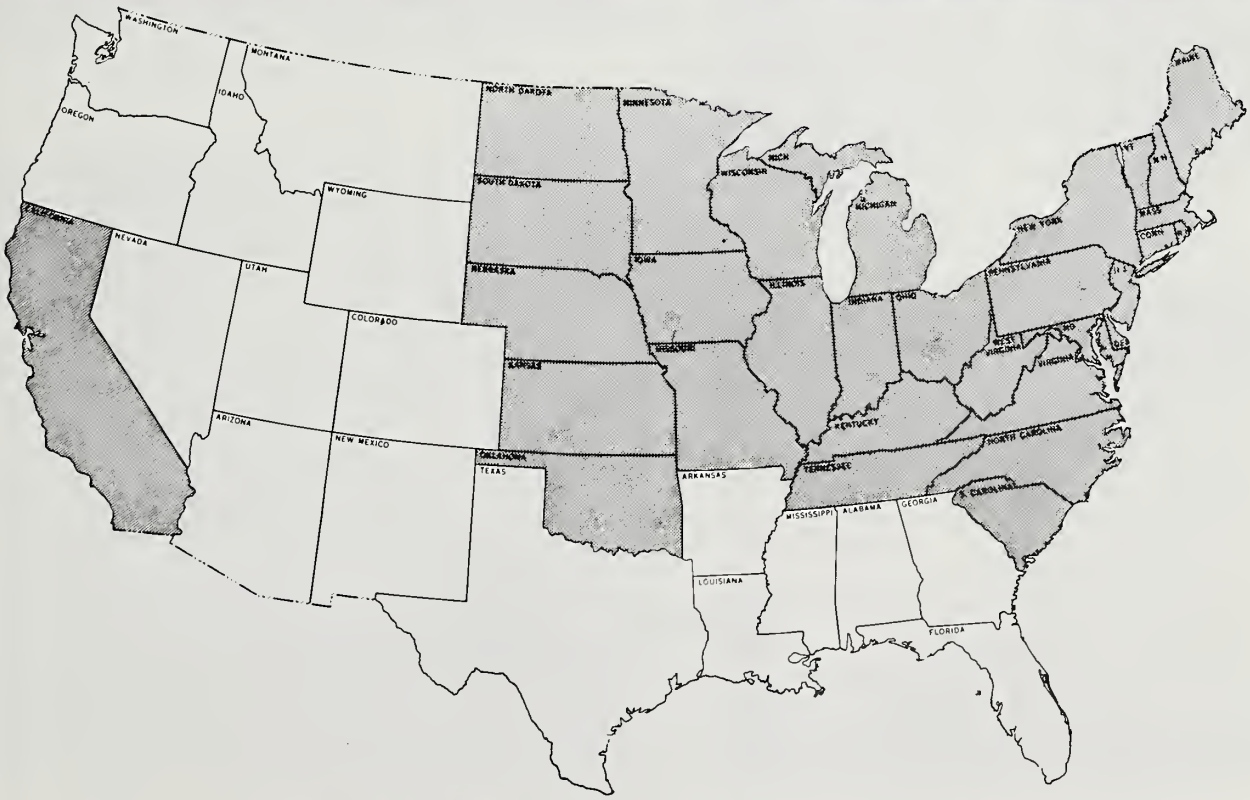

Figure 4-2.—*Diplodia pinea* occurs in shaded states and in Hawaii.

Figure 4-4.—New shoots of Austrian pine killed by *Diplodia pinea*.

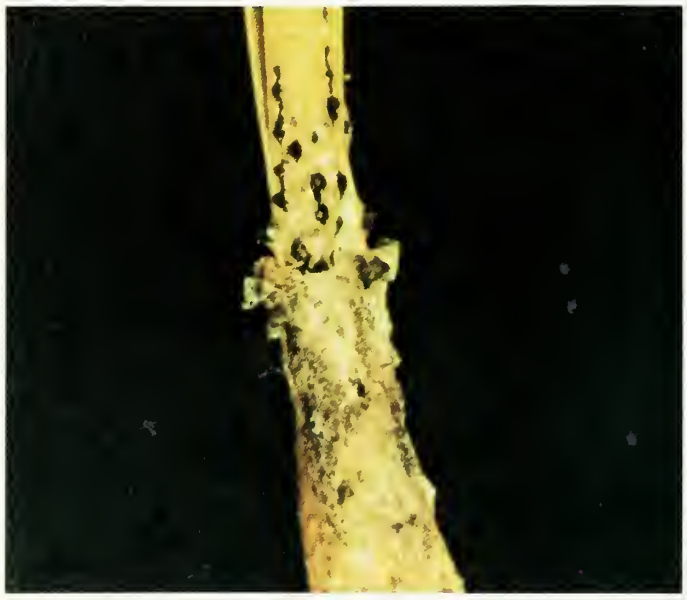

Figure 4-5.—Fruiting bodies (pycnidia) of *Diplodia pinea* numerous at base of Austrian pine needle.

Figure 4-6.—Fruiting bodies (pycnidia) on an Austrian pine seed cone infected by *Diplodia pinea* (left); uninfected cone (right).

Figure 4-7.—Spores of *Diplodia pinea*.

Figure 4-8.—Severe damage to Austrian pines caused by *Diplodia pinea*.

Figure 4-9.—Top of Austrian pine tree killed by *Diplodia pinea*.

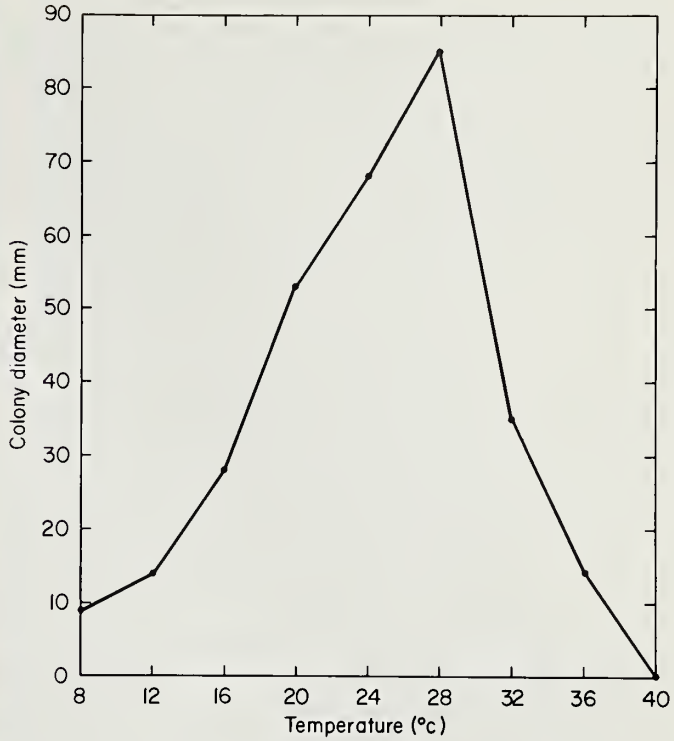

Figure 4-11.—Effect of temperature on colony diameter of *Diplodia pinea* grown on potatoe dextrose agar for 48 hours.

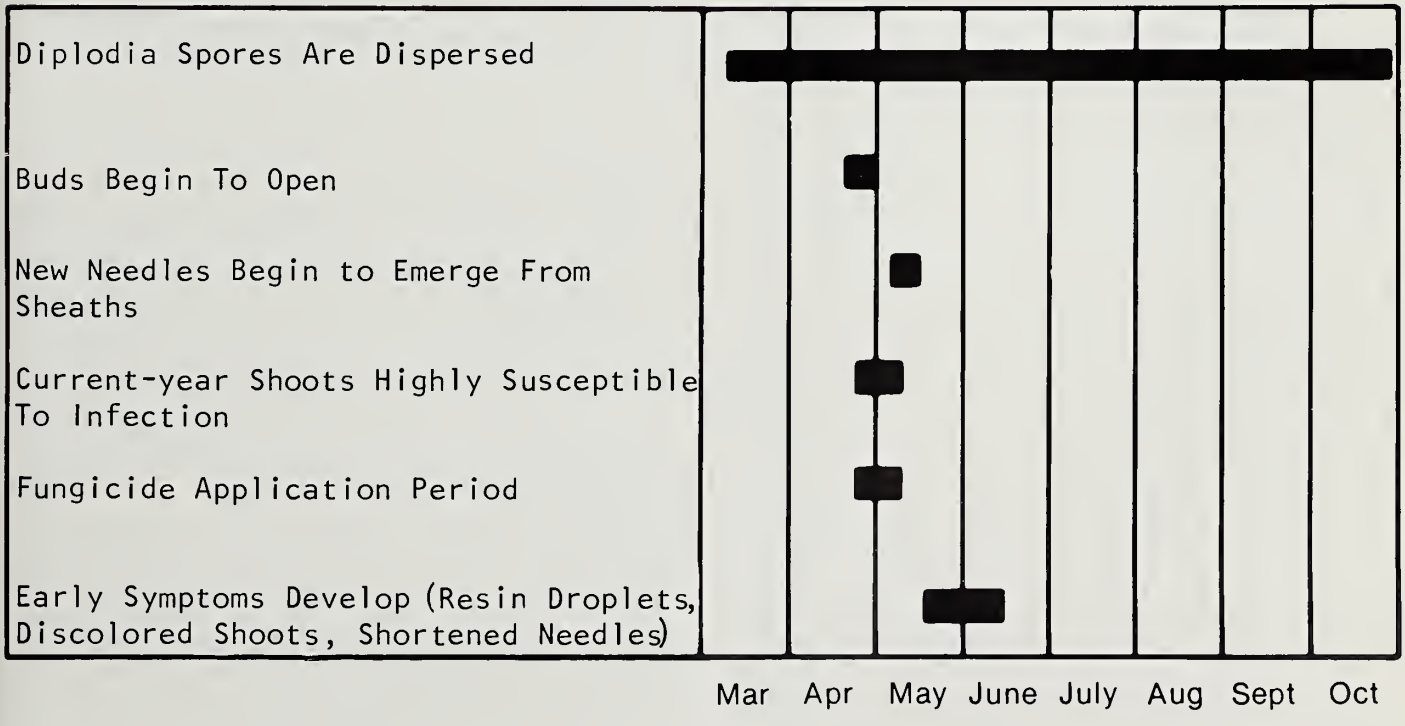

Figure 4-10.—Schedule for developing programs for control of Diplodia blight.

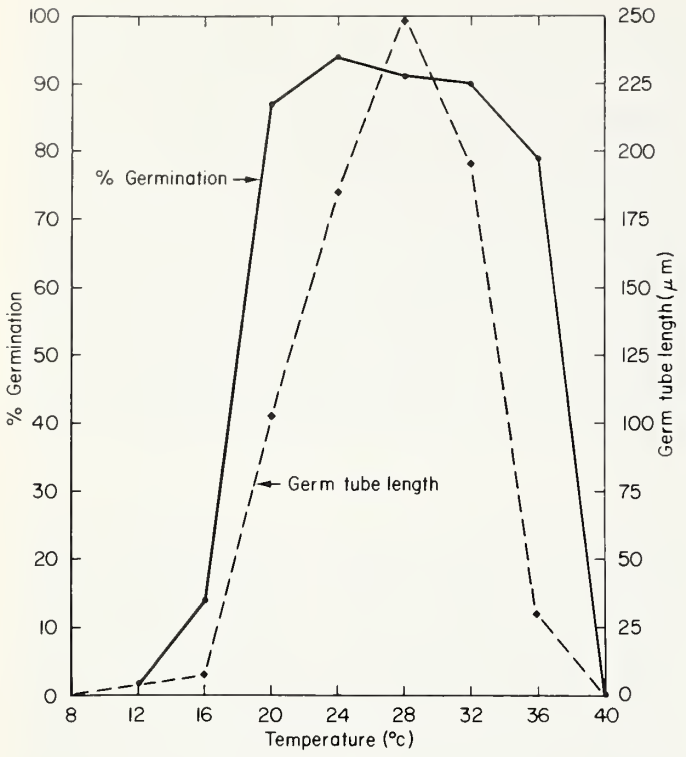

Figure 4-12.—Effect of temperature on germination of *Diplodia pinea* spores incubated for 4 hours on water agar.

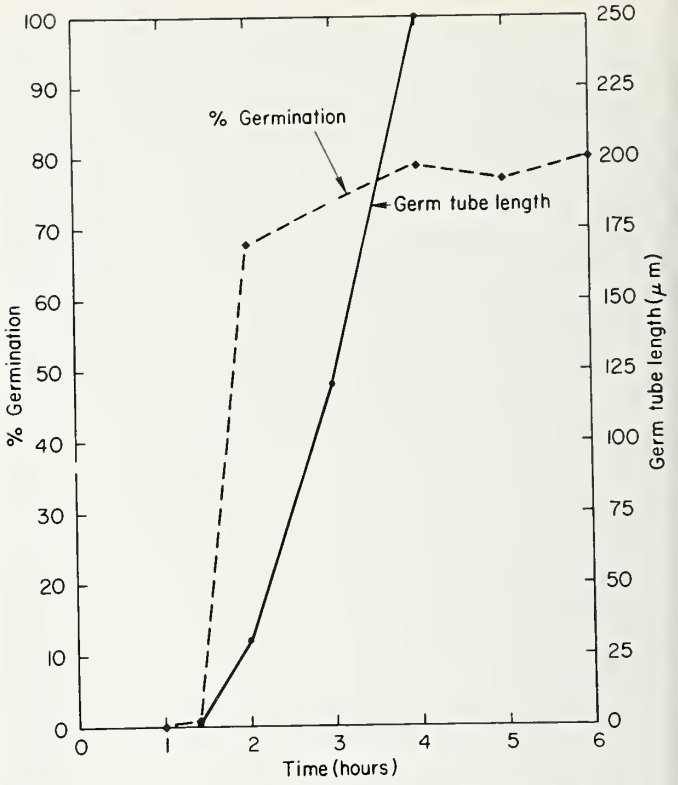

Figure 4-13.—Germ tube length and percent germination of *Diplodia pinea* spores incubated for various periods of time, at 26°C, on water agar.

5. Western Gall Rust of Pines

Infection of pines by the western gall rust fungus *Peridermium harknessii* results in formation of branch and stem galls (figs. 5-1, 5-2) and sometimes cankers. The quality of wood in gall-bearing stems is reduced, and the stems are subject to wind breakage.

Ponderosa and Scots pines are most frequently damaged by this fungus in the Great Plains; however, the fungus has a wide host range, including lodgepole, jack, and mugo pines.

Surveys of all natural ponderosa pine stands in Nebraska, in the early 1920's, revealed that gall rust was present in all the stands. There is a high percentage of infected trees, in natural ponderosa stands, in the Pine Ridge area, in northwestern Nebraska, and in natural stands and plantings of ponderosa in the Black Hills of South Dakota.

Gall rust has seldom been detected in shelterbelts established in the Great Plains. The exception to this is in North Dakota, where plant pathologists at North Dakota State University have found gall rust in a high percentage of shelterbelts.

Diagnosis

The distinctive galls which develop on infected trees make it easy to detect western gall rust (fig. 5-3). This rust can be confused with eastern gall rust (*Cronartium quercuum*) because the galls of both are similar. The two fungi can be distinguished by differences in germ tubes developing from spores formed in galls. Determining these differences requires germination of the spores and comparison of growth rate and branching of germ tubes, a task best handled by specialists.

Life Cycle

The galls on pines produce spores in the spring. These spores are dispersed primarily by wind. The main period of spore dispersal is in May and early June. The spores infect newly developing shoots of pines. Thus this is a pine to pine rust; that is, infection of pines does not require a non-pine (alternate) host. In contrast, eastern gall rust is a pine-oak rust; spores produced in galls on pines infect oak leaves. The spores produced on the oaks infect pines.

Galls seldom are formed the year trees are infected. Most are formed the second year. They are first evident in July or August. The galls formed in July and August of the second year produce spores the following spring. The surface of the galls breaks open, exposing the bright-colored spores (fig. 5-4, 5-5). The spores are wind-dispersed in May and June.

The time between initial infection and sporulation is approximately 24 months. In rare instances, galls form on shoots during the same growing season that the shoots are infected; these galls then produce spores the following spring.

Galls continue to produce spores each year. They cease only when the stem tissue bearing galls dies.

The best time to check for infection is in August or later, when galls resulting from infection the previous year are evident. However, if a planting is being checked for the purpose of reducing the amount of inoculum (spores) of the rust fungus, the checks should be made before spores are dispersed in the spring. In most areas, this would mean making the checks before the last week in April. Checks for presence of rust could be made in early summer, but they would reveal only old galls that have already sporulated.

Infection of Nursery Seedlings

Because the spores are dispersed by wind, pines far from a source of spores (infected tree) may become infected. Pine seedlings in nursery beds, located 200 m from old infected pines, became infected. The level of infection was low (less than 0.5%) in the seedling beds. This nursery has routinely checked for presence of rust in pines near the nursery; infected trees or the branches bearing galls are removed.

Infection of nursery stock, even a low percentage of stock, is serious because spores will develop on and be dispersed to other trees in outplantings. Although nurseries certainly do not distribute seedlings which have galls, some infected seedlings will not have galls on them when they are distributed for planting. Seedlings which are infected the last year they are in the nursery will not have galls on them when they are distributed for spring planting. Galls will develop on such seedlings during July and August of the first year they are planted. Typically these seedlings will have galls at the base of the main stem (fig. 5-6).

Rate of Spread in Outplantings

Gall rust was detected in several experimental plantings, established in 1968, which contained from 50 to 80 geographic sources of ponderosa pine. The initial source of infection was obviously nursery stock, because the trees had galls at the base of main stems, and there were no infected pines near the planting sites.

It was determined that approximately 1 seedling in 1,000 was infected in the nursery. Next, the rate of disease spread within the plantings was determined. During annual examinations through the spring of 1977, determinations were made of the number of trees infected and their distance and direction from epicenters (trees initially infected in the nursery). A total of 879 trees were infected; by 1977, 58% and 76% of these were within 26 m and 38 m, respectively, of trees that had been infected in the nursery.

Infected trees were uniformly distributed around epicenters; incidence of infected trees was as high in one direction from the epicenter as incidence in other directions.

There was a high level of new infections in 1973 but not in 1972 or 1974. Some 53% of all diseased trees were initially infected in 1973, and 56% of the total number of galls were on 1973 internodes. Since spores were available for infection during the three years, the

yearly variation in infection was probably due to weather conditions (moisture, temperature) being more favorable for infection in 1973.

Control

The level of rust infection was reduced from 4% to less than 0.5% by removing branches containing galls or entire infected trees located near the nursery. This sanitation procedure is carried out each year before galls sporulate in the spring.

During the course of a study on spread of the rust fungus within young, experimental pine plantings, all galls observed were removed. The approximate number of trees in two of the plantings was 5,000 and 8,000. Removing galls no doubt reduced the level of infection; however it was impossible to find every gall no matter how carefully trees were checked. In a planting of 3,000 pines, one tree was found that had been initially infected in the nursery. This tree was removed and a careful check of all trees in the planting was made for 3 successive years. The considerable increase in the number of infected trees in the planting indicates that not all of the galls were found. Perhaps in a very small planting, rust, if detected early, could be eradicated by gall removal.

The best way to ensure that gall rust does not develop in Great Plains plantings is to use nursery stock grown where infection does not occur, that is stock grown where there are no infected pines near the nursery. There are no fungicides that currently are EPA registered for control of this rust fungus in the Great Plains.

Physiology and Morphology

Readers are referred to the bulletin by R. S. Peterson (1967) for information on the physiology and morphology of *P. harknessii*.

Figure 5-1.—Galls on Scots pine infected by *Peridermium harknessii*.

Figure 5-2.—Large gall on stem of Scots pine.

Figure 5-3.—Galls of various shapes on ponderosa pine branches infected by the western gall rust fungus.

Figure 5-5.—Gall with surface removed, exposing masses of *Peridermium harknessii* spores.

Figure 5-4.—Surface of gall splitting open in mid-May revealing orange spores of *Peridermium harknessii.*

Figure 5-6.—Galls on lodgepole pine seedlings infected in the nursery with *Peridermium harknessii.*

6. Phomopsis Blight of Junipers

Phomopsis juniperovora has been a threat to nurserymen producing junipers since the 1880's (fig. 6-1). This fungus disease is common in the Great Plains, from South Dakota to Texas and eastward to the Atlantic Coast (fig. 6-2). Losses have been especially severe in seedling beds of *Juniperus virginiana* and *J. scopulorum* and in the production of grafted selections of these and other juniper species. *Cupressus arizonica* seedlings have been seriously damaged by this fungus in some southern states. Several other species in the Cupressaceae family are susceptible.

Diagnosis and Life Cycle

Phomopsis initially infects foliage, then spreads to and kills stem tissues. Newly developing needles are especially susceptible while they are still in the yellowish green stage; after needles develop to a normal, deep green, they are not susceptible. Small, yellowish spots appear on needles of *J. virginiana* and *J. scopulorum* within 3 to 5 days after infection. The fungus permeates young needles and rapidly invades young stem tissues (fig. 6-3). As a result, terminals and branches become light green, then red-brown and, finally, ashen gray. When a side shoot is infected, the fungus progresses to the main stem, which it may girdle if the stem is less than 1 cm in diameter (fig. 6-4). The portion of the seedling above the girdled area then dies. Lesions on larger stems frequently develop into cankers, but the stems are not girdled. The fungus does not spread far below cankers. Survival is poor if infected bareroot stock is outplanted. A comparison of blighted and non-blighted trees outplanted in eastern Nebraska revealed that survival of blighted stock after 2 years was 30% less than non-blighted stock.

Phomopsis fruiting bodies (pycnidia), with viable spores, may develop within 3 to 4 weeks after seedlings become infected. Well developed pycnidia are usually found on needles and stems that have dried and turned ashen gray. The small, dark pycnidia are at first embedded in needles and stems, but later they erupt through the epidermis (fig. 6-5). The spores are extruded in whitish yellow tendrils. Two types of spores (alpha and beta) develop in the same or different pycnidia (figs. 6-6, 6-7). Alpha spores are colorless, one-celled, ellipsoid, contain two oil globules, and commonly are 7.5-10 by 2.2-2.8 μm. Beta spores are colorless, one-celled, filamentous, slightly curved, and 20.2-26.9 by 1 μm.

Frequently, only alpha spores are found in pycnidia. This makes disease identification difficult, because there are some saprophytic fungi found on junipers that produce spores similar to Phomopsis alpha spores. However, these spores usually do not have the two characteristic oil globules. Specialists can readily distinguish these saprophytes from Phomopsis by using such cultural media as potato dextrose agar or malt extract agar. On these cultural media, *P. juniperovora* produces a characteristic deep yellow coloration, usually accompanied by appearance of orange-red crystals (Fig. 6-8).

Another fungus, *Cercospora sequoiae*, which causes a blight of junipers and other species in the Cupressaceae family, can be easily distinguished from Phomopsis blight. Cercospora infection originates on older needles of lower branches and spreads upward and outward, whereas Phomopsis infection starts on new needles on branch tips and advances inward.

Damage from drought can be confused with Phomopsis blight. In both cases, tips of branches may be killed. However, the demarcation between green and dead tissue will be sharp in Phomopsis-blighted seedlings but gradual in seedlings affected by drought.

Damage from the lesser cornstalk borer can be distinguished from Phomopsis blight by the straw color of the dead tops and by the feeding wounds on the lower stem and taproot.

Recently, *Kabatina juniperi* has become a problem in juniper outplantings and in production of grafted junipers. This fungus causes symptoms similar to those caused by Phomopsis. However, there are differences in spore size and fruiting bodies between this fungus and Phomopsis, which a specialist can recognize.

Control

Fungicides are needed for effective control in seedling beds (fig. 6-9). Mercury fungicides have been effective against Phomopsis; however, there are now restrictions against use of mercury fungicides. Most of the non-mercury fungicides in general use are not effective against Phomopsis. Bordeaux mixture is frequently recommended for control, but is ineffective. Several relatively new fungicides have been tested in recent years in a South Dakota nursery. The tests have shown that benomyl, which is EPA registered for use against Phomopsis, is effective.

Protective fungicides, such as benomyl, must be applied regularly to protect new growth. In Great Plains nurseries, fungicide is typically applied weekly, beginning with seedling emergence and ending after the last flush of growth in the fall. Because the fungus initially infects only foliage at the yellowish-green stage, infection is highest during periods of new growth. In most Great Plains nurseries, juniper seedlings have a flush of growth in the spring and another flush in late summer or early fall. Some of the worst losses occur during late flushes of growth. In some Great Plains nurseries, there is a period in midsummer when little or no infection occurs, because there is little or no new growth. The frequency of fungicide applications possibly could be reduced during this period. However, more tests are necessary, because 3-0 *J. virginiana*, grown under irrigation in a South Dakota nursery, were infected throughout the growing season whenever branches bearing Phomopsis pycnidia were placed into the nursery beds.

Moderate temperatures (16° to 26°C) and rain are conducive to infection. However, applying fungicides only after rain is impractical, because Phomopsis

spores need only about 7 hours to germinate, enter, and infect seedlings. This period would be too short in many cases to prepare equipment and apply spray in time to prevent infection. In addition, tests in South Dakota have shown that, if benomyl is applied weekly at 0.5 pound per acre, residue on new growth is sufficient to prevent disease development, even after periods of moderate rainfall (12 to 22 mm).

Removing infected seedlings from beds (roguing) can reduce the amount of infection, because the disease apparently progresses slowly from infected to uninfected areas of nursery beds, if only a few infected trees are present.

In South Dakota, infected branches bearing Phomopsis pycnidia were placed into uninfected 2-0 *J. virginiana* beds when new growth began in the spring. One month later, all new infection was within 1 foot of the branches that had been placed into the beds. Infected seedlings should be rogued only when they are dry, to minimize the spread of spores. Rogued seelings should be immediately placed in a container, such as a plastic bag, to minimize chances of spores being dispersed. The infected seedlings then should be disposed of carefully. Weekly roguing of all seedlings with dying foliage, coupled with benomyl sprays at 7- to 10-day intervals, has provided complete control of Phomopsis blight at the Big Sioux State Conifer Nursery, Watertown, S. Dak., since 1974.

Sowing juniper seed adjacent to beds containing juniper stock should be avoided, if possible, so as to reduce chances of spores from infected older stock infecting new seedlings. Junipers or other hosts of this fungus should not be used in nursery windbreaks or in landscape plantings on nursery ground, because they may be a source of inoculum (spores) for nursery stock. Such trees are likely to be infected extensively if pruning results in development of juvenile foliage.

Poorly drained areas also should be avoided, because losses are often greater where water tends to pool (fig. 6-10). Overhead sprinkler irrigation should be terminated in time for free water to evaporate from foliage before dusk. Because shading frames increase the time that water remains on foliage, they should not be used unless absolutely necessary.

Phomopsis blight is particularly difficult to control in the highly moist conditions required for production of juniper grafts. Watering can disperse spores present on scions or understocks, and the long periods when free water is present on the foliage favors infection. Under these highly moist conditions, the most effective fungicides are apt to fail. The nurseryman has to obtain a balance in the moisture regime so that graft production will be successful, and Phomopsis infection will not occur. This is especially difficult if grafts are produced in enclosures in which temperatures are moderate and water is applied at frequent intervals by mist systems.

Some nurseries have abandoned production of highly susceptible cultivars (i.e. *Juniperus sabina tamariscifolia*) because of continued high losses. *Phomopsis juniperovora* can cause unsightly junipers in landscapes but seldom kills established trees (fig. 6-11). The objective of Phomopsis control in landscapes is to reduce the unsightly conditions. Infection of outplanted junipers can be severe during unusually moist growing seasons, but infection is usually moderate in most years. Infection can be reduced by restricting pruning and shearing to periods when the resulting flush of new growth will occur during the drier part of the growing season. Junipers are sometimes pruned after the first flush of growth in the spring; this can result in a new flush of growth in early summer, when rain may be plentiful. Such new growth is especially vulnerable to infection.

Fungicides can be used for control of *P. juniperovora* in landscapes, although tests have not been made to define the most effective and economical spray schedules. Frequent applications of fungicide would be impractical in outplantings, but applying fungicide during the early and late flushes of growth is practical and probably would be adequate in most years.

Physiology and Morphology

Spores are extruded from pycnidia in tendrils or globules. Globules usually form when free water is present on tissues containing pycnidia. Temperature affects the rate at which spores are extruded from pycnidia. On branches kept in the dark at 100% relative humidity, spores were extruded within: 23 hours at 24°C and 28°C; 30 hours at 20°C; 45 hours at 16°C; and 93 hours at 12°C. Spores were not extruded at either 32° or 36°C, after 165 hours. Spores were extruded at similar rates whether pycnidia-bearing tissues were incubated in light or dark at 24°C or 28°C and 100% relative humidity.

Spores in tendrils are tightly bound by a matrix. Considerable agitation is required to disperse spores in tendrils placed in water. Spores were not dispersed after 7 hours when tendrils were placed in water and not agitated.

Alpha spores germinated over the temperature range 12° to 32°C when incubated for 18 hours on water agar (fig. 6-12). Spores germinated at 8°C after incubation for 34 hours but not after 18 hours. More than 60% of the spores germinated over the range 12° to 32°C.

Germ tube growth was greatest at 26°C (fig. 6-12). Germ tubes were evident 4 hours after incubation started. Germ tubes developed slowly from 4 to 12 hours; from 12 to 28 hours, development was rapid and nearly linear with time (fig. 6-13). Germ tube development was better on water agar and cornmeal sucrose agar than on potato dextrose agar. Germ tubes grew more rapidly (44 μm versus 10 μm) and percent germination was higher (100% versus 15%) when spores were incubated on water agar than when incubated in distilled water, after 18 hours, at 24°C.

Percentage spore germination and germ tube lengths were similar whether spores germinated in the dark or in incandescent or fluorescent light.

Spores obtained from tendrils that had been exposed to temperatures of 1°. 10° and 24°C for 24 hours ger-

minated 100%; exposure at 33-43°C resulted in 80% germination and germ tubes about one-half as long (24 μm) as at the lower temperatures. Spores subjected to -22°C in water or in tendrils still germinated.

Spores which had been hydrated, then dried at temperatures ranging from 4° to 54°C, still germinated. Increase in temperature or length of drying period decreased percent germination and germ tube lengths.

Growth of *P. juniperovora* on agar media was optimum at 24-26°C (fig. 6-14). The rate of growth from 3 to 9 days was linear with time. Growth was greater on potato dextrose agar and cornmeal sucrose agar than on prune agar and malt extract agar (fig. 6-15). Colony diameters of 27 isolates, after 6 days, at 24°C, on cornmeal sucrose agar ranged from 5.3 to 8.0 cm. Seventeen isolates had colony diameters between 7 and 8 cm. Growth was as rapid in the dark at it was in the light (1075 lux).

All isolates developed a yellow color in advance of the colony periphery. Color intensity varied among isolates. The color intensity of all isolates was the same whether grown in light or dark. Orange-red crystals formed in all media. The major constituent of the orange-red crystals is a tetrahydroquinone (Wheeler, Wheeler, and Peterson 1975) which was previously isolated by Stoessl (1967) and named by him altersolanol A.

Pycnidia were not produced in abundance on these media, even when cultures were incubated in the light. Excellent pycnidial production was obtained using asparagine agar with sucrose as the carbon source.

Asparagine Agar

KH_2PO_4	1.0 g
$MgSO_4.7H_2O$	0.5 g
Asparagine	2.3 g
Agar (Difco Noble Agar)	20.0 g
Sucrose	10.0 g
Distilled water	1.0 l

Light (280 lux) was required for pycnidial production. Production of pycnidia with fertile spores was extensive with 3 to 4 weeks incubation at 24°C. Production was better with sucrose than with glucose, fructose, lactose, or mannitol. Production using sucrose at 10 g/l or 20 g/l was better than using 2 g/l sucrose.

Infection of Seedlings under Controlled Conditions

Seedlings of *J. virginiana* and *J. scopulorum*, inoculated with spore suspensions and incubated at temperatures ranging from 16° to 32°C (100% relative humidity), were readily infected. Infection at 12°C was very light. Intensity of infection was greater at 24°C to 28°C than at other temperatures.

Seedlings became infected after only 7 hours incubation at 24°C and 100% relative humidity. The amount of infection was similar whether inoculated plants were incubated in the dark or in the light (2-day continuous fluorescent, 12,900 lux). After incubation for 2 days at 24°C, disease development was enhanced by higher post-incubation temperatures (28-32°C). The level of infection with 7 to 9 hours incubation was only 21% of level of infection with 24 hours incubation. Infection levels after 1, 2, or 3 days of incubation were similar; however, the level of infection was 50% less when both *J. virginiana* and *J. scopulorum* were incubated for 4 days.

The information on inoculations with *P. juniperovora* is being used to evaluate resistance among progenies of 86 select eastern redcedar trees located in the Great Plains. Resistance to *P. juniperovora* is present within eastern redcedar (fig. 6-16). Research is seeking to determine if there is genetic resistance which can be utilized to reduce the impact of this fungus on eastern redcedar seedlings in nurseries.

Figure 6-1.—Damage by *Phomopsis juniperovora* to second-year eastern redcedar seedlings.

Figure 6-2.—*Phomopsis juniperovora* occurs in shaded states.

Figure 6-3.—Stem and branch tips of second-year eastern redcedar seedlings killed by *Phomopsis juniperovora.*

Figure 6-4.—Typical damage caused by *Phomopsis juniperovora* —dead reddish tops with ashen grey tissues where stem girdled.

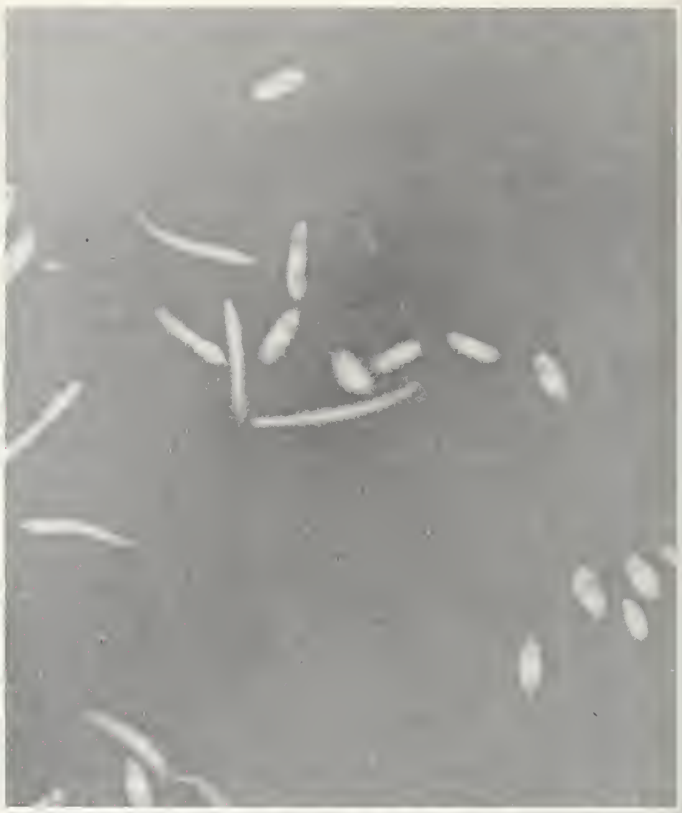

Figure 6-6.—Alpha (short) spores and beta (long) spores of *Phomopsis juniperovora*. Alpha spores have two oil globules.

Figure 6-5.—Black fruiting bodies (pycnidia) of *Phomopsis juniperovora* on stem and leaf of eastern redcedar.

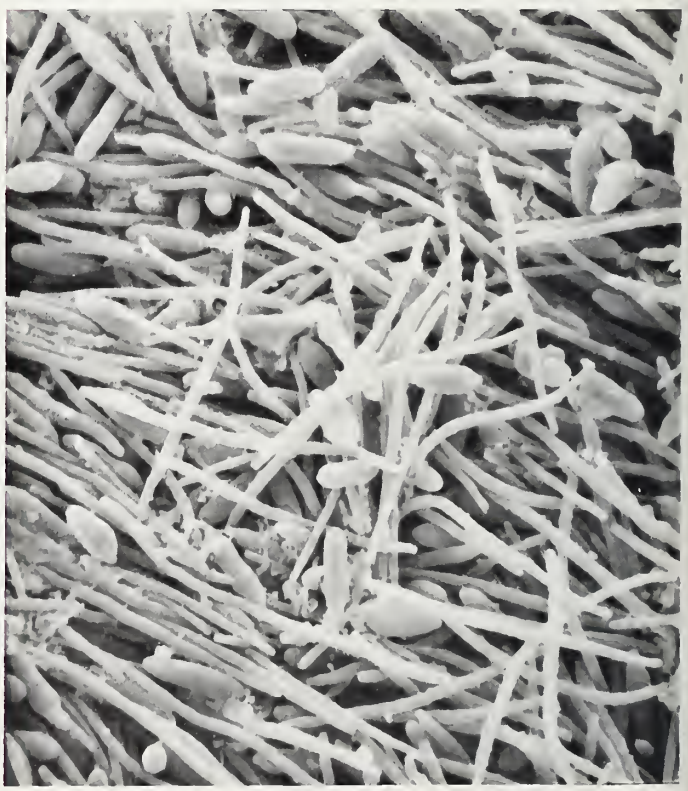

Figure 6-7.—Alpha and beta spores of *Phomopsis juniperovora* magnified 2000 times.

Figure 6-8.—Typical yellow coloration of *Phomopsis juniperovora* colony.

Figure 6-9.—Control of Phomopsis blight of eastern redcedar seedlings by a protective fungicide (foreground); unsprayed seedlings in background.

Figure 6-10.—Severe infection by *Phomopsis juniperovora* in a poorly drained (low) area in the nursery.

Figure 6-11.—Eastern redcedar in a 7-year-old planting with branch tips infected by *Phomopsis juniperovora*.

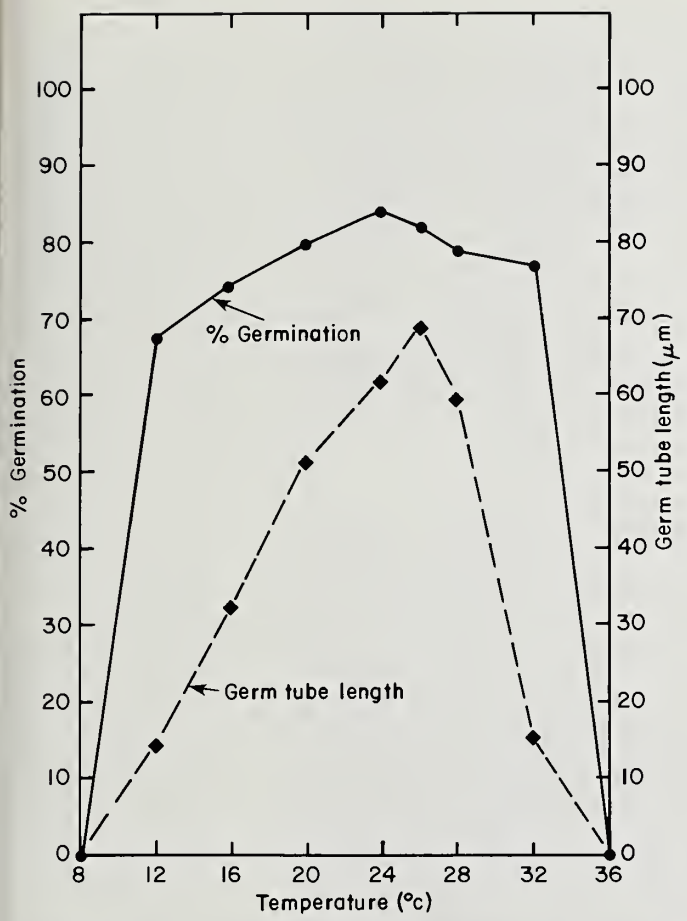

Figure 6-12.—Effect of temperature on germination of spores of *Phomopsis juniperovora* incubated for 18 hours on water agar.

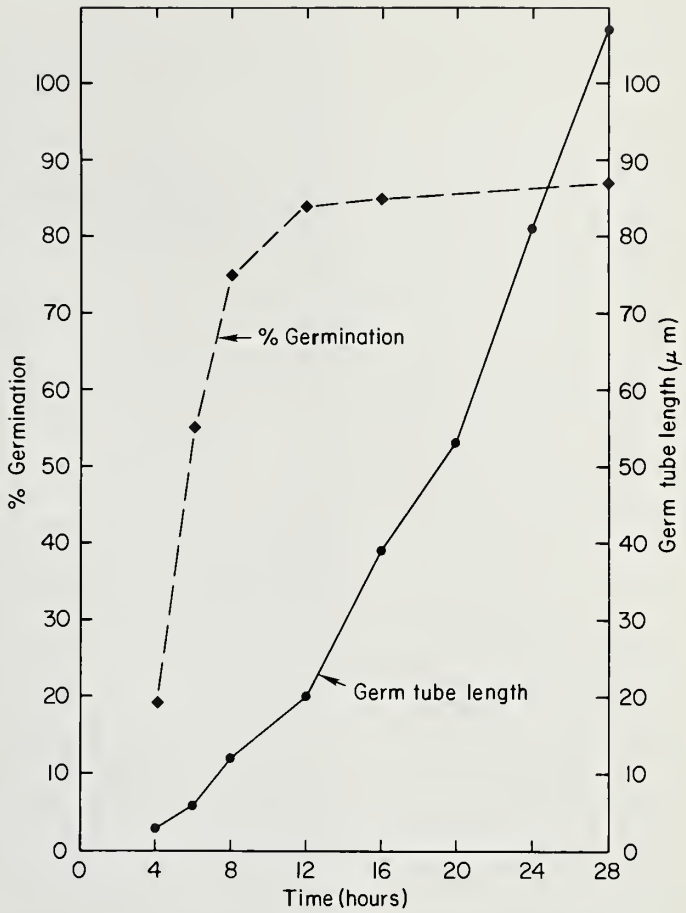

Figure 6-13.—Germ tube length and percent germination of *Phomopsis juniperovora* spores incubated for various periods of time, at 24°C, on water agar.

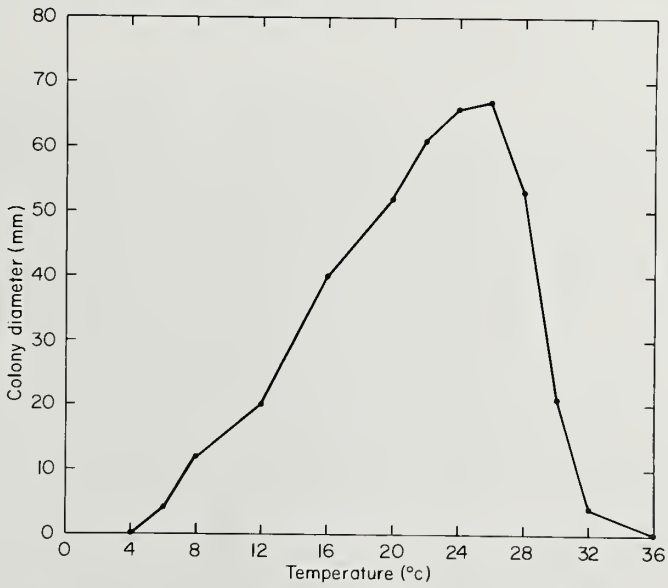

Figure 6-14.—Effect of temperature on colony diameter of *Phomopsis juniperovora* grown for 6 days on corn meal sucrose agar.

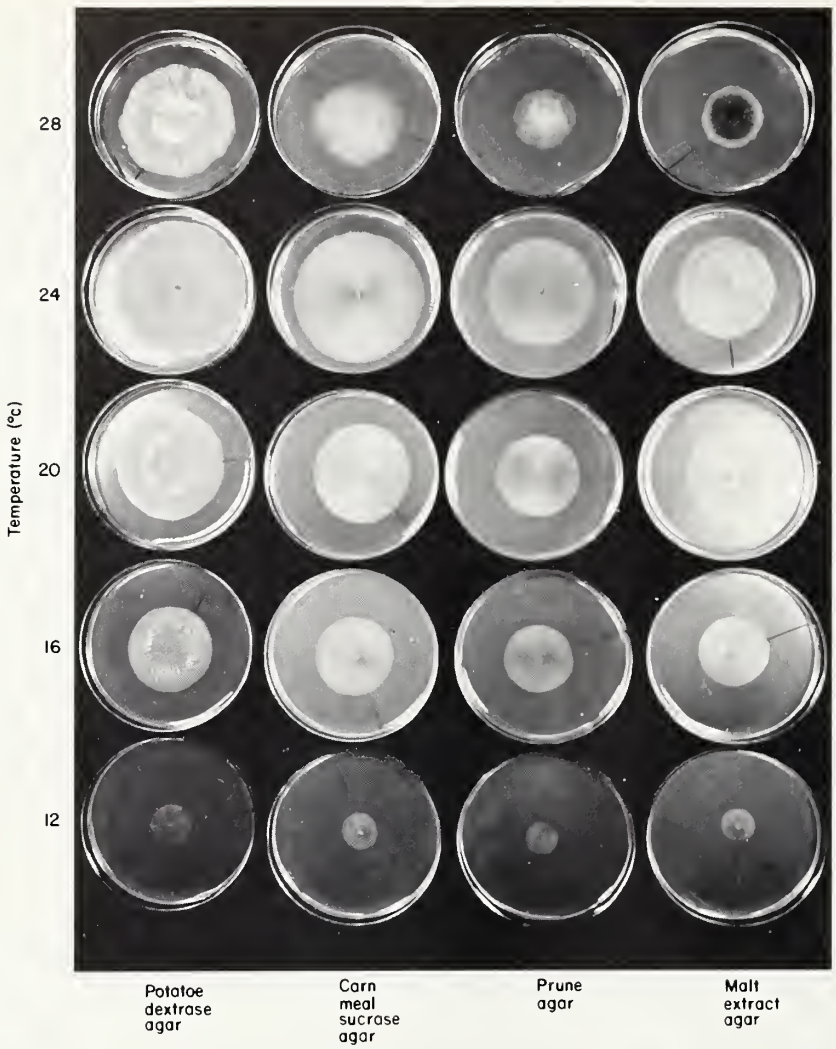

Figure 6-15.—Effect of temperature and cultural media on appearance and growth of *Phomopsis juniperovora* colonies after 6 days.

Figure 6-16.—Resistant eastern redcedar trees adjacent to a tree severely infected by *Phomopsis juniperovora*.

7. Cercospora Blight of Junipers

Junipers are damaged by the fungus *Cercospora sequoiae* and the related fungus *Cercospora sequoiae* var. *juniperi*. In the Great Plains, *C. sequoiae* var. *juniperi* has severely damaged *Juniperus virginiana* and *J. scopulorum* in well established windbreaks and other plantings (fig. 7-1). Several successive seasons of infection can kill trees. The distribution of these two fungi in the United States is shown in figure 7-2.

This fungus has not been a threat to production of juniper seedlings in the Great Plains; it is seldom found on nursery seedlings. However, Cercospora infection has been commonly observed in nurseries on grafted selections of junipers, particularly selections of *J. scopulorum* that have been kept in the nursery for 5 or 6 years.

Diagnosis

Cercospora blight is easier to diagnose than Phomopsis and Kabatina blights of junipers. The branches of Cercospora infected trees usually will be devoid of foliage near their bases but will have healthy foliage on their tips (fig. 7-1); whereas, the branches of Phomopsis and Kabatina infected trees will have dead tips.

Juniper foliage has been grouped into three types: (1) whip leaves characteristic of long shoot growth on the ends of secondary and tertiary branches; (2) spur leaves characteristic of short (spur) branches; and (3) juvenile leaves characteristic of seedlings.

Early symptoms are bronzed tips of leaves on spur shoots. Subsequently these leaves become entirely bronzed, then necrotic. Commonly, all leaves of a branchlet are affected. Infected foliage on branchlets usually dies in late September.

Affected branchlets drop from trees in October and November, resulting in the typical appearance of diseased trees—the extremities of the branches bear healthy green foliage and the inner crown is devoid of foliage. Following severe infection, juvenile foliage commonly develops on branches which previously have had only spur and whip foliage.

Life Cycle

Spores of the fungus are dispersed from late April through October. Dispersal may not be abundant until late May or June, and no spores are dispersed during rainless periods. There is little or no long-distance dispersal of spores; no spores were collected in traps located within 6 feet of severely infected trees. There is considerable yearly variation in the amount of infection. Infection has been severe when rainfall during the growing season was at or above average. Infection was slight during the drought years of 1975 and 1976 in eastern Nebraska. Moisture is required for spores to disperse and germinate and for the fungus to penetrate foliage.

Junipers in eastern Nebraska are first infected in early to midsummer. Initial infection occurred during the period July 14 to 28, 1971 and June 21 to July 5, 1972. On spur leaves, symptoms first were observed August 8, 1971 and July 19, 1972. The period between initial infection and first appearance of symptoms is between 2 and 3 weeks.

On juvenile foliage, symptoms were first evident July 19, 1972; by September 26, most of the infected leaves were necrotic. By October 3 necrosis was extreme. This indicates that the disease develops more rapidly in juvenile leaves than in spur leaves. Also, both current-year and previous-years' juvenile leaves became infected, whereas only the previous-years' spur leaves became infected.

Fruiting bodies (sporodochia) (figs. 7-3, 7-4) resulting from current-season's infection were observed in spur foliage September 26, 1974. They sporulated readily when incubated at 24°C and 100% relative humidity for 18 hours. In 1973, sporodochia first were found on spur foliage collected in September. Sporodochia first were found September 5, 1972 on juvenile leaves infected that year.

Tips of secondary and tertiary branches on 10-year-old *J. virginiana* were free of infection for an average distance of 45 cm. Measurements of the distance between the disease-free ends of these branches and the limit of infection of 1971 revealed that the disease had extended an average distance of 28 cm along the branches in 1972. Lack of infection on tips may be a result of less moisture on the outermost foliage because of more rapid drying. Whip foliage which develops on branch tips also may be resistant to infection.

Trees planted in north-south rows had much more infection on the west side than on the east side. The longer persistence of moisture from dew or evening rains on the west side probably accounts for the higher levels of infection.

The disease develops more rapidly in *J. scopulorum* than in *J. virginiana* (fig. 7-5). In a shelterbelt containing a row of both species, 91% of the *J. scopulorum* died, compared to 41% of the *J. virginiana*. The fungus killed more *J. scopulorum* (fig. 7-6) and *J. monosperma* than *J. virginiana*, in a conifer test planting near Mapleton, Iowa.

Control

Because trees with whip and spur foliage were not infected before late June (and then only previous years' foliage became infected), a highly persistent fungicide applied before late June, theoretically, could protect trees with spur and whip foliage for the entire season. Because of fungicide weathering, however, an additional application in late July usually is required.

Because both current-year and previous years' juvenile foliage become infected, juniper trees containing juvenile leaves would require additional fungicide applications to protect newly developing juvenile leaves. Bordeaux mixture (8-8-100) provided a high degree of control in tests.

Park managers using control procedures outlined (fig. 7-7) have controlled the Cercospora blight fungus on *J. scopulorum* and on *J. virginiana* since 1973. The timing of fungicide applications shown in figure 7-7

was developed from tests in eastern Nebraska. Timing should be modified slightly in other areas—earlier application in southern locations, and later applications in northern locations.

Cercospora blight is found more frequently in new plantings of *J. scopulorum* than in new plantings of *J. virginiana*. Where Cercospora may be a problem, it would be better to plant *J. virginiana*.

Variation in resistance to cultivars of these two junipers species has not been systematically evaluated. To determine whether there is usable genetic resistance to Cercospora, a test planting of progenies from 158 select trees in the Great Plains, was made in eastern Nebraska, in 1980. Results from this test should be available in the early 1980's.

Physiology and Morphology

Growth of the fungus in malt extract broth was greatest at 24°C (fig. 7-8).

More than 90% of spores germinated on water agar within 24 hours, over the range 16° to 28°C; no spores germinated at 8°C or 32°C (fig. 7-9). Germ tube growth was greatest at 24-26°C (fig. 7-9). Germination began within 6 hours at 24°C, and percent germination was near maximum within 16 hours (fig. 7-10). Percent germination and germ tube lengths were similar whether spores were incubated (24°C) in the dark or in the light.

The age of cultures from which spores were obtained strongly influenced spore germination and germ tube growth. After incubation for 24 hours at 24°C, germination and germ tube lengths for spores from 7-day-old cultures were 91% and 47 μm, from 10-day-old cultures 57% and 22 μm, and from 14-day-old cultures 16% and 10 μm, respectively. Spores were produced in quantity on carrot-leaf decoction agar (Kilpatrick and Johnson, 1956) when incubated under continuous fluorescent light (624 lux).

Spores are cylindrical, olive brown, slightly spiny, 1-7 septate (mostly 5-6) (figs. 7-11, 7-12). Spores ranged from 16.5-69.5 μm by 2.0-3.6 μm, and averaged 40.8 by 3.1 μm. Spores are borne at the apex of the conidiophores, and on new growing points which develop just below and to one side of the previously developed spore.

Figure 7-1.—Typical appearance of Rocky Mountain juniper severely damaged by *Cercospora sequoiae* var. *juniperi*.

Figure 7-2.—Geographic distribution of *Cercospora sequoiae* and *Cercospora sequoiae* var. *juniperi*. *Cercospora sequoiae* (dots); *Cercospora sequoiae* var. juniperi (gray); both (black).

Figure 7-3.—Dark fruiting bodies of *Cercospora sequoiae* var. *juniperi* on Rocky Mountain juniper foliage.

Figure 7-4.—Closeup of fruiting bodies of *Cercospora sequoiae* var. *juniperi* showing fuzzy gray appearance of conidia and conidiophores.

Figure 7-5.—Damage by *Cercospora sequoiae* var. *juniperi*; damage is more severe to Rocky Mountain juniper (left) than to eastern redcedar (right), in an eastern Nebraska windbreak.

Figure 7-6.—A Rocky Mountain juniper planting in western Iowa destroyed by *Cercospora sequoiae* var. *juniperi.*

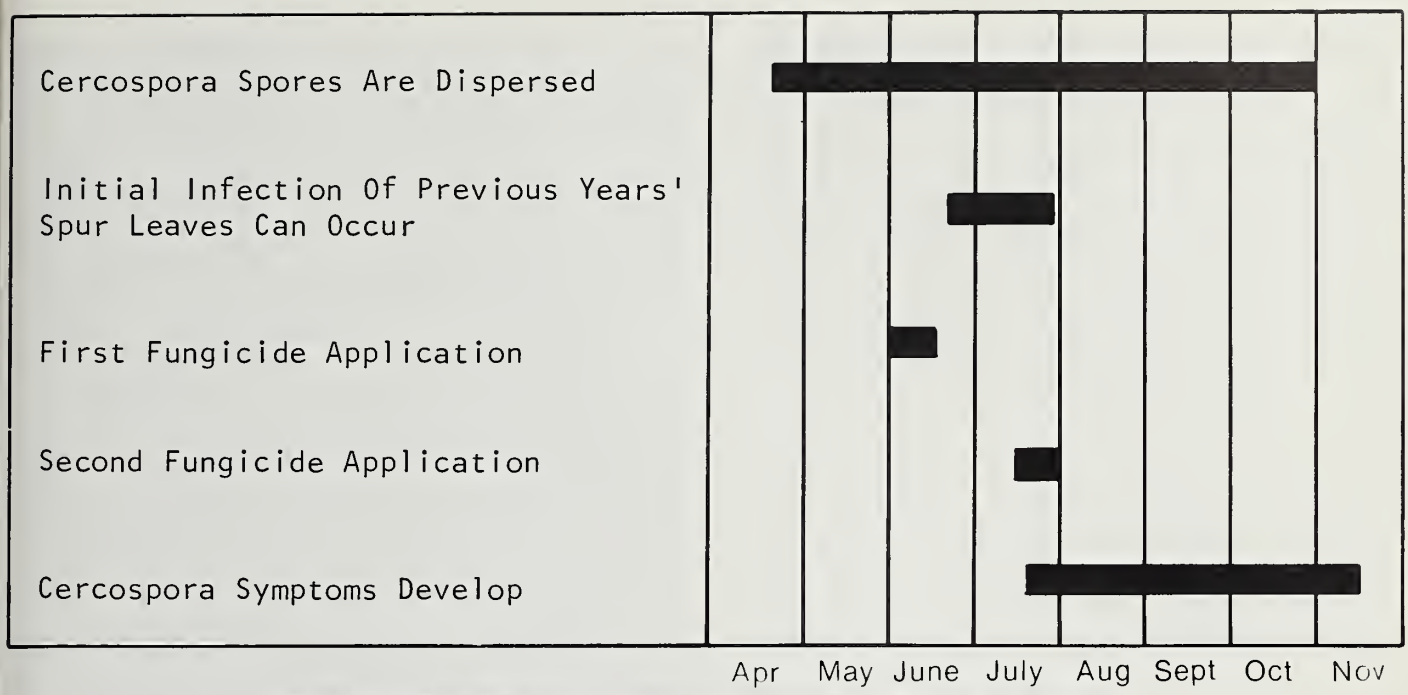

Figure 7-7.—Schedule for developing programs for control of Cecospora blight.

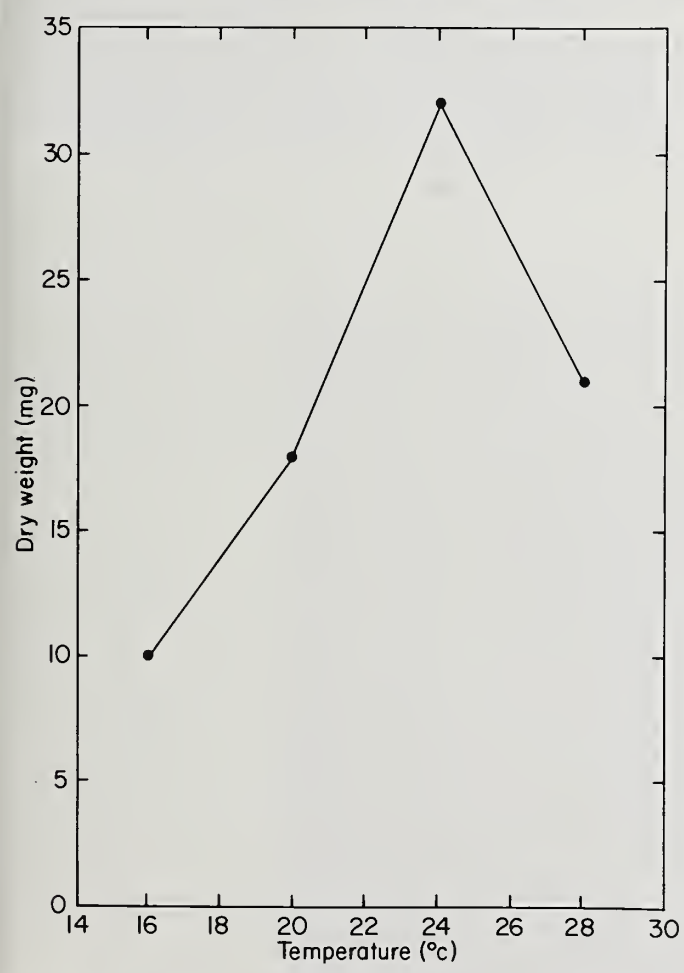

Figure 7-8.—Effect of temperature on growth of *Cercospora sequoiae* var. *juniperi* in a liquid culture medium, after 14 days.

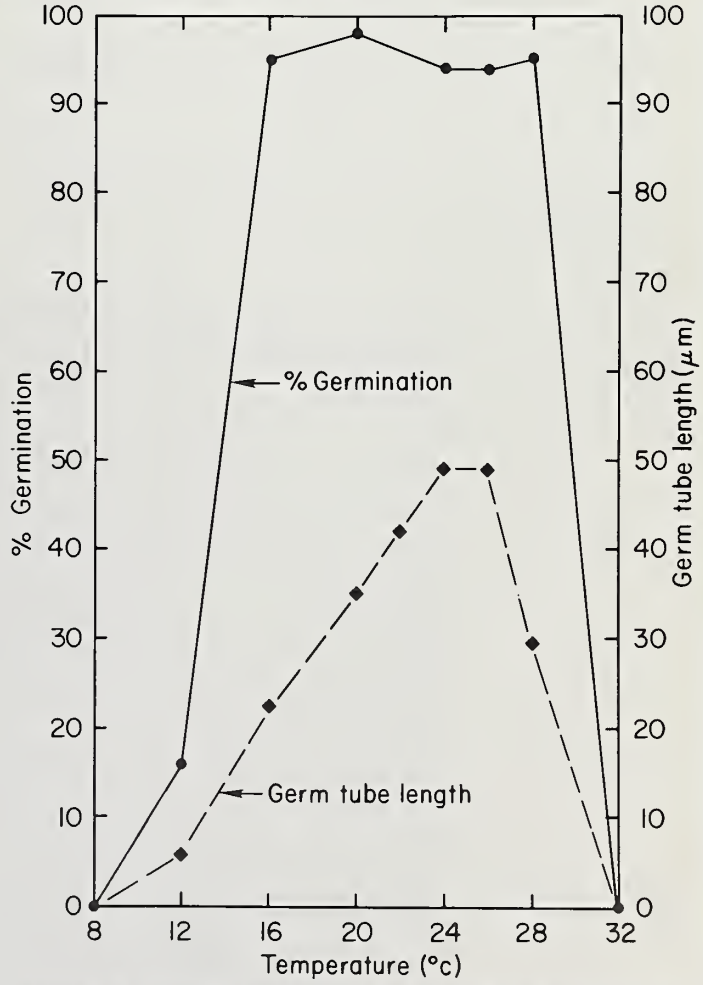

Figure 7-9.—Effect of temperature on germination of *Cercospora sequoiae* var. *juniperi* spores incubated for 24 hours on water agar.

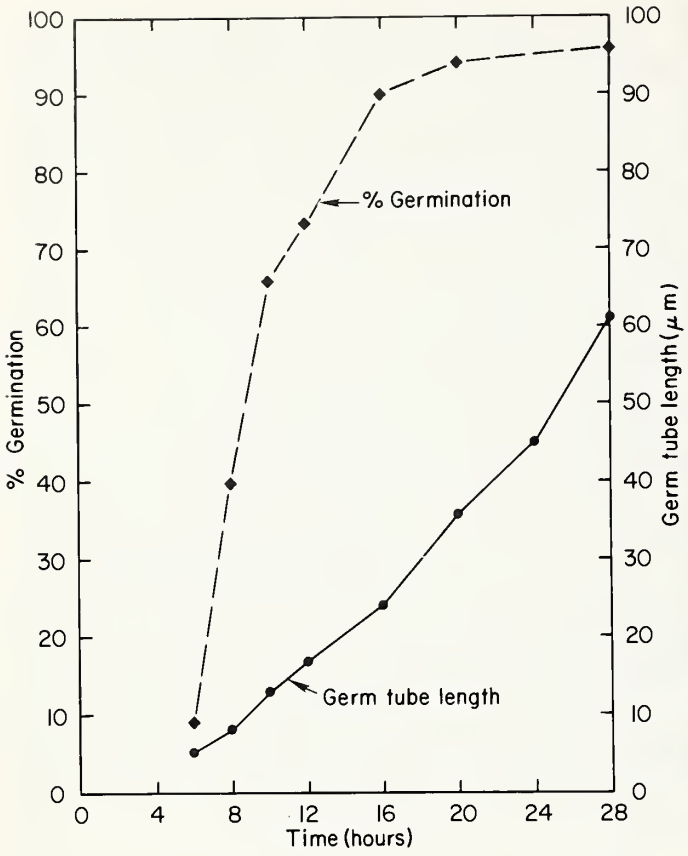

Figure 7-10.—Germ tube length and percent germination of *Cercospora sequoiae* var. *juniperi* spores incubated for various periods of time, at 24°C, on water agar.

Figure 7-12.—Spiny surface of spores of *Cercospora sequoiae* var. *juniperi*. (X 3750)

Figure 7-11.—Spores of *Cercospora sequoiae* var. *juniperi* showing cross walls (septations).

8. Kabatina Blight of Junipers

For several years, branch tips of eastern redcedar in Nebraska have been damaged by an unknown agent in plantings (fig. 8-1). Recently, the fungus *Kabatina juniperi* was found to be the cause of this damage. *Juniperus scopulorum* is also infected.

There is very little information on the geographic distribution of this fungus in the Great Plains or in the rest of the United States; it has been found in Nebraska, Indiana, Wisconsin, and New Hampshire.

Diagnosis and Life Cycle

The symptoms of Kabatina blight are first evident in the spring at the time when juniper foliage turns deep green. Foliage on branch tips infected with *K. juniperi* turn yellow-brown instead of green (fig. 8-2).

The average length of dieback of diseased branches of eastern redcedar was 128 ± 50 mm. An average of 10 mm of this dieback was covered with *K. juniperi* fruiting bodies (acervuli). Average twig diameter at the base of the dieback was 1.6 ± 0.4 mm.

The fruiting bodies of *K. juniperi* are usually present at the base of discolored foliage in a zone of sunken tissue (fig. 8-3). The fruiting bodies are numerous in April and May, but decline in number throughout the summer. Hyaline, ellipsoid, one-celled spores (4.5-8.0 μm by 2.3-3.0 μm) are formed singly at the tips of septate sporophores (fig. 8-4).

The damage caused by *K. juniperi* resembles damage caused by *Phomopsis juniperovora* (compare figs. 8-1 and 6-11). The time of symptom development is helpful in distinguishing these two blights. Kabatina blight symptoms develop before new growth begins in the spring, whereas Phomopsis blight symptoms develop anytime during the growing season.

The period of infection is unknown. Tissues that become discolored in early spring probably are infected the previous year, because there are few spores available for infection at time of discoloration, and the weather conditions at time of discoloration probably would be unfavorable for infection.

Artificial inoculation of eastern redcedar seedlings revealed that only seedlings with wounded foliage became infected. Scanning electron microscope photographs revealed that the fungus grew profusely around wounds and entered leaves through wounds.

The cause of the wounds observed on foliage of eastern redcedar in eastern Nebraska plantings is unknown. However, two unidentified insects, capable of wounding foliage, have been found in eastern redcedar plantings infected with *K. juniperi*.

Control

No tests for control of this fungus have been conducted. Control tests are planned which will include both fungicides and insecticides, applied alone and in combination, at different times. The tests will be arranged so that the time or times when infection occurs can be determined.

Physiology and Morphology

The optimum temperature for growth of *K. juniperi* is 24°C (fig. 8-5). The optimum temperature for germination of spores of *K. juniperi* on water agar is 24°C; this temperature is also optimum for germ tube growth (fig. 8-6).

Germination began between 8 and 12 hours when spores were incubated on water agar at 24°C; about 80% of the spores had germinated with 20 hours of incubation (fig. 8-7).

Artifically inoculated eastern redcedar seedlings, incubated for 5 days, at 100% relative humidity, at 16°, 20°, 24°, or 28°C, became infected if foliage had been wounded before inoculation. Wounded, inoculated seedlings incubated for 24 hours, at 100% relative humidity, at 24°C, also became infected.

Figure 8-1.—Eastern redcedar damaged by *Kabatina juniperi*.

Figure 8-2.—Tips of eastern redcedar branches killed by *Kabatina juniperi*.

Figure 8-3.—Fruiting bodies of *Kabatina juniperi* on eastern redcedar.

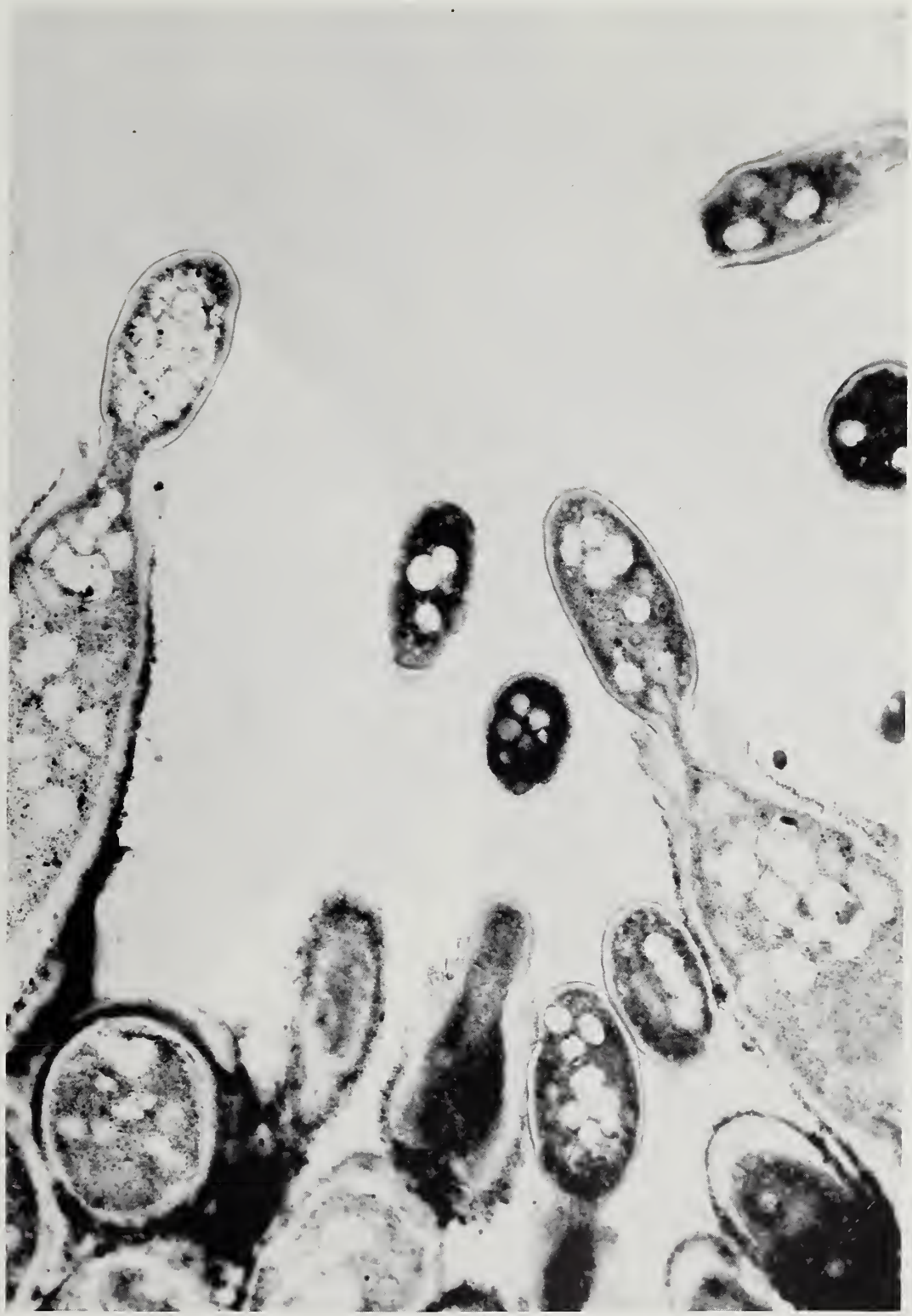

Figure 8-4.—Spores and sporogenous cells of fruiting body of *Kabatina juniperi.* (X 10,000)

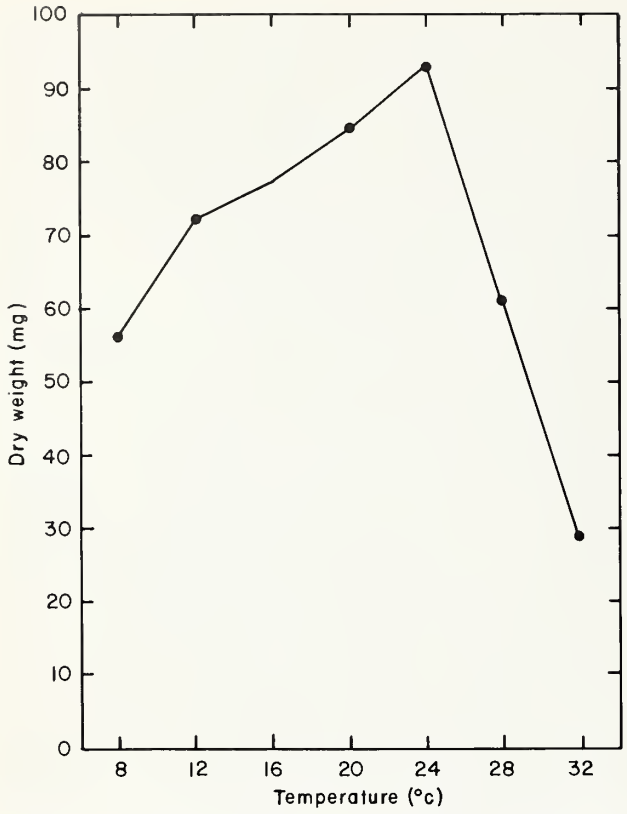

Figure 8-5.—Effect of temperature on growth of *Kabatina juniperi* in a liquid culture medium, after 7 days.

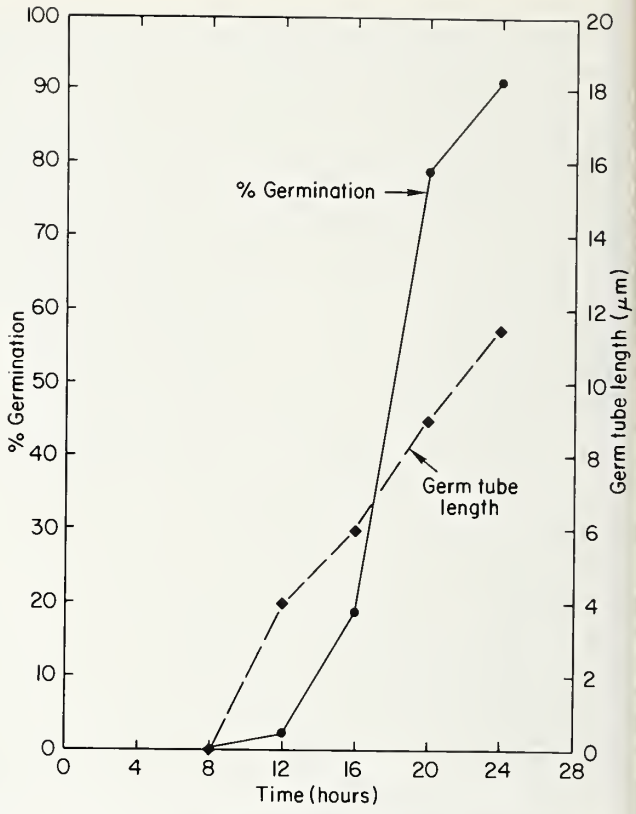

Figure 8-7.—Germ tube length and percent germination of *Kabatina juniperi* spores incubated for various periods of time, on water agar, at 24°C.

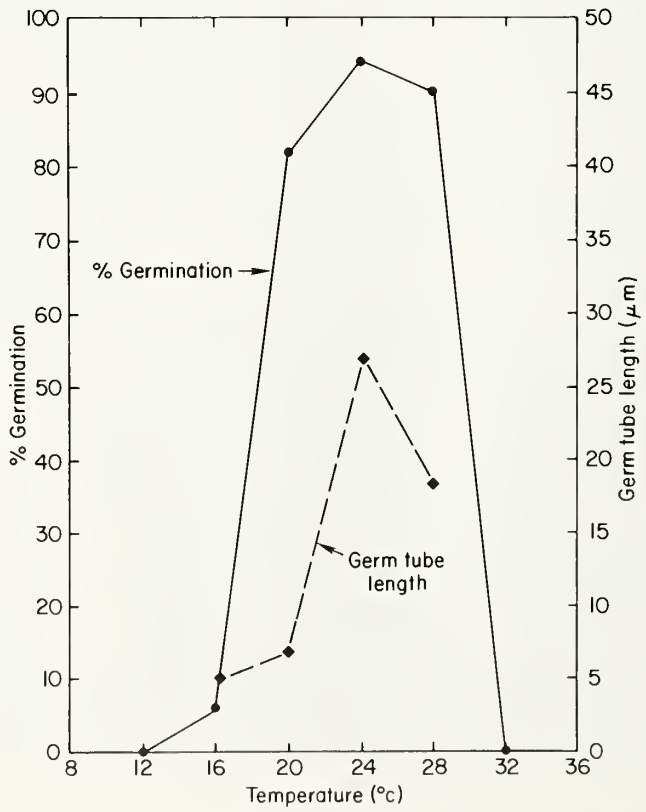

Figure 8-6.—Effect of temperature on germination of *Kabatina juniperi* spores incubated for 24 hours on water agar.

9. Root Lesion Nematode Damage to Junipers and Pines

Nematodes infecting tree roots can cause large economic losses in tree nurseries. The losses occur because the growth of seedlings is reduced to the extent that they are not salable. In addition, most states prohibit the sale of nematode-infected trees. The root lesion nematode *Pratylenchus penetrans* has a very wide host range which includes many of the plant species commonly used for cover crops in tree nurseries. In the early 1960's, tree nurseries in the Great Plains were surveyed for damage by nematodes. Root lesion nematodes were encountered more frequently than other kinds of plant pathogenic nematodes.

Diagnosis and Life Cycle

Damage by root lesion nematodes was particularly severe in a forest tree nursery in north-central Nebraska. Eastern redcedar seedlings in beds adjacent to eastern redcedar windbreaks were in low vigor (fig. 9-1). There was no obvious damage to the aerial parts of junipers in the windbreaks. However, an examination of roots of the windbreak trees and roots of the adjacent seedlings revealed that they were infected with the root lesion nematode *P. penetrans*.

Before root lesion nematodes were found, several of the windbreaks had been removed because the nursery beds were being reoriented. The soil mounded in the windbreaks was spread out so that the nursery blocks would be relatively level.

Subsequently, eastern redcedar and Rocky Mountain juniper seeds were sown in the areas where windbreaks had been removed. At that time, seedlings were kept in beds for 2 years then transplanted for an additional year's growth before being distributed for field planting. Towards the end of the first growing season, there were some areas where seedling growth was much reduced. By the end of the second growing season the seedlings in these areas were stunted and in low vigor (fig. 9-2).

In every case, the low vigor seedlings had high infestations of root lesion nematodes. Root lesion nematodes are endoparasites—they enter roots and feed within them. As a consequence, many of the roots die, which accounts for stunted and low vigor seedlings. The seedlings whose roots were being killed formed new roots, which also became infected by the nematodes. These new roots were much larger and fleshier than normal roots. In checking for presence of root lesion nematodes, it was easier to use the fleshy roots because there were fewer kinds of nematodes than in the older roots.

Initially, damage was observed only on juniper roots. However, a check of roots of other seedlings revealed that all conifers growing in the nursery were hosts for this nematode. These trees were Austrian pine, ponderosa pine, jack pine, Colorado blue spruce, Black Hills spruce, eastern redcedar, and Rocky Mountain juniper.

The nematode problem in this nursery was increased when seedlings from affected areas were transplanted; root lesions nematodes were introduced into the transplant area of the nursery, which previously had been free of root lesion nematodes.

Control

After nematodes were detected, several control experiments were conducted to determine the most effective soil fumigant. The fumigant needed had to be one that would move into and out of the soil rapidly. This was necessary because both seeding and transplanting were done in the spring. Furthermore, all space in the nursery was being utilized during the growing season.

For effective fumigation, soil temperatures need to be at least 13°C. Because temperatures remained low early in the season, there was little time between lifting of seedlings and either seeding or transplanting.

Tests showed that methyl bromide was effective, and moved into and out of the soil rapidly. Therefore, seeding or transplanting could be done soon after fumigation (figs. 9-3, 9-4).

Soon after nematodes became a problem, the nursery, in an effort to reduce costs, explored ways of eliminating transplanting. Two methods were tried: (1) growing seedlings in the same beds for 2 years but at lower densities and with higher soil fertility, and (2) growing seedlings in the same beds for 3 years.

Evaluations showed that seedlings grown in beds that had been fumigated with methyl bromide were essentially free of nematode damage after two growing seasons; but if they remained in the beds for 3 years, the root lesion nematode populations increased to the level where the third-year seedlings were severely damaged.

In the fall, areas free of seedlings were commonly sown to rye or oats to reduce soil movement by wind. Unfortunately, both rye and oats were hosts of *P. penetrans* (fig. 9-5). The nursery now uses cover crops that are not hosts of *P. penetrans*.

This nursery produces high quality stock, but only because they use soil fumigants to control root lesion nematodes. The wide host range of the nematode, plus the fact that, even with the most effective fumigant, the nematode populations increase to damaging levels after 2 years, dictates that soil fumigation must remain a standard practice in this nursery (fig. 9-6). Soil fumigation has the added benefits of controlling weeds and reducing losses caused by damping-off disease fungi.

Figure 9-1.—Root lesion nematode damage to eastern redcedar seedlings, adjacent to an eastern redcedar windbreak that was heavily infected by root lesion nematodes.

Figure 9-2.—Extensive root lesion nematode damage to eastern redcedar seedlings in an area where an eastern redcedar windbreak had been removed.

Figure 9-3.—Effectiveness of soil fumigation with methyl bromide (Dowfume MC-2) in controlling root lesion nematodes.

Figure 9-4.—Root lesion nematode (*Pratylenchus penetrans*) damage to eastern redcedar seedlings in a small area where soil was not fumigated with methyl bromide.

Figure 9-5.—A cover crop of oats severely damaged (foreground) by root lesion nematodes; tall, deep, green oats in background are not infected.

Figure 9-6.—Fumigation of nursery beds with methyl bromide to control root lesion nematodes.

Selected References

1. Dothistroma Needle Blight of Pines

Bassett, C., Margaret Buchanan, R. T. Gallagher, and R. Hodges. 1970. A toxic difuroanthraquinone from *Dothistroma pini*. Chemistry and Industry in New Zealand 52:1659-1660.

Funk, A., and A. K. Parker. 1966. *Scirrhia pini* n. sp., the perfect state of *Dothistroma pini* Hulbary. Canadian Journal of Botany 44:1171-1176.

Gadgil, P. D. 1967. Infection of *Pinus radiata* needles by *Dothistroma pini*. New Zealand Journal of Botany 5:498-503.

Gadgil, P. D. 1975. Effect of temperature and leaf wetness period on infection of *Pinus radiata* by *Dothistroma pini*. New Zealand Journal of Forestry Science 4:495-501.

Gadgil, P. D. 1977. Duration of leaf wetness periods and infection of *Pinus radiata* by *Dothistroma pini*. New Zealand Journal of Forestry Science 7:83-90.

Gadgil, P. D., and G. Holden. 1976. Effect of light intensity on infection of *Pinus radiata* by *Dothistroma pini*. New Zealand Journal of Forestry Science 6:67-71.

Garcia, Julio, and Jochen Kummerow. 1970. Infection of Monterey pine graftings with *Dothistroma pini*. Plant Disease Reporter 54:403-404.

Gibson, I. A. S. 1972. Dothistroma blight of *Pinus radiata*. Annual Review of Phytopathology 10:51-72.

Gibson, I. A. S. 1974. Impact and control of Dothistroma blight of pines. European Journal of Forest Pathology 4:89-100.

Gilmour, J. W., and A. Noorderhaven. 1971. Influence of time of application of cuprous oxide on control of Dothistroma needle blight. New Zealand Journal of Forestry Science 1:160-166.

Hulbary, Robert. 1941. A needle blight of Austrian pine. Illinois Natural History Survey Bulletin 21:231-236.

Ivory, M. H. 1967. Spore germination and growth in culture of *Dothistroma pini* var. *keniensis*. Transactions of the British Mycological Society 50:563-572.

Ivory, M. H. 1972. Infection of Pinus radiata foliage by *Scirrhia pini*. Transactions of the British Mycological Society 59:365-375.

Parker, A. K. 1972. Artificial inoculation of *Pinus radiata* with *Scirrhia (Dothistroma) pini*: Effect of relative humidity and temperature on incubation. Phytopathology 62:1160-1164.

Peterson, Glenn W. 1965. Dothistroma needle blight of Austrian pine: Infection and control. Plant Disease Reporter 49:124-126.

Peterson, Glenn W. 1966. Penetration and infection of Austrian and ponderosa pines by *Dothistroma pini*. Phytopathology 56:894-895 [abstract].

Peterson, Glenn W. 1967. Dothistroma needle blight of Austrian and ponderosa pines: Epidemiology and control. Phytopathology 57:437-441.

Peterson, Glenn W. 1967. Dothistroma needle blight of pines in North America. Proceedings 14th Congress International Union of Forestry Research Organizations [Munich, 1967] 5:269-278.

Peterson, Glenn W. 1969. Growth of germ tubes of *Dothistroma pini* conidia positively directed toward stomata of Austrian and ponderosa pine needles. Phytopathology 59:1044 (abstract)

Peterson, Glenn W. 1973. Infection of Austrian and ponderosa pines by *Dothistroma pini* in eastern Nebraska. Phytopathology 63:1060-1063.

Peterson, Glenn W. 1975. Dothistroma needle blight: A problem in production of landscape pines. American Nurseryman 141 (12):11, 94, 95, 96.

Peterson, Glenn W. 1975. Dothistroma needle blight of pines. p. 72-75. *In* Forest Nursery Diseases in the United States. U. S. Department of Agriculture, Agriculture Handbook 470, 125 p. Washington, D. C.

Peterson, Glenn W. 1976. Some reflection on biological studies of needle diseases. USDA Forest Service Research Note RM-323, 4 p. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

Peterson, Glenn W., and David A. Graham. 1974. Dothistroma blight of pines. USDA Forest Service, Forest Pest Leaflet 143, 5 p. Washington, D. C.

Peterson, Glenn W., and George M. Harvey. 1976. Dispersal of *Scirrhia (Dothistroma) pini* conidia and disease development in a shore pine plantation in western Oregon. Plant Disease Reporter 60:761-764.

Peterson, Glenn W., and Ralph A. Read. 1971. Resistance to *Dothistroma pini* within geographic sources of *Pinus nigra*. Phytopathology 61:149-150.

Peterson, Glenn W., and James A. Walla. 1978. Development of *Dothistroma pini* on and within needles of Austrian and ponderosa pines in eastern Nebraska. Phytopathology 68:1422-1430.

Sheridan, J. E., and Chea Chark Yen. 1970. A note on the effect of temperature and relative humidity on the germination of conidia of a New Zealand isolate of *Dothistroma pini* Hulbary. New Zealand Journal of Botany 8:658-660.

Thyr, B. D., and C. Gardner Shaw. 1964. Identity of the fungus causing red band disease on pines. Mycologia 56:103-109.

Walla, James A., and Glenn W. Peterson. 1976. *Dothistroma pini* and *Diplodia pinea* not affected by surface wax of pine needles. Plant Disease Reporter 60:1042-1046.

2. Brown spot needle blight of pines

Kais, A. G. 1971. Dispersal of *Scirrhia acicola* spores in southern Mississippi. Plant Disease Reporter 55:309-311.

Kais, Albert G. 1975. Brown spot needle blight. p. 69-71. *In* Forest nursery diseases in the United States. U. S. Department of Agriculture, Agriculture Handbook 470, 125 p. Washington, D. C.

Kais, Albert G. 1975. Environmental factors affecting brown-spot infection on longleaf pine. Phytopathology 65:1389-1392.

Kais, Albert G. 1975. Fungicidal control of *Scirrhia acicola* on longleaf pine seedlings. Plant Disease Reporter 59:686-688.

Kais, Albert G. 1977. Influence of needle age and inoculum spore density on susceptibility of longleaf pine to *Scirrhia acicola*. Phytopathology 67:686-688.

McGrath, W. Thomas, Allen J. Prey, and Frank S. Morse. 1972. Brown spot needle blight of Scotch pine Christmas trees in Wisconsin: Sporulation and control. Plant Disease Reporter 56:99-101.

Nicholls, Thomas H., Darroll D. Skilling, and George W. Hudler. 1973. *Scirrhia acicola* in Scotch pine Christmas tree plantations. Plant Disease Reporter 57:55-59.

Phelps, W. R., Albert G. Kais, and Thomas H. Nicholls. 1978. Brown-spot needle blight of pines. U. S. Department of Agriculture, Forest Service, Forest Insect and Disease Leaflet 44, 8 p. Washington, D. C.

Siggers, Paul V. 1944. The brown spot needle blight of pine seedlings. U. S. Department of Agriculture, Technical Bulletin 870, 36 p. Washington, D. C.

Skilling, Darroll D., and Thomas H. Nicholls. 1974. Brown spot needle disease—biology and control in Scotch pine plantations. USDA Forest Service Research Paper NC-109, 19 p. North Central Forest Experiment Station, St. Paul, Minn.

Wolf, Frederick A., and W. J. Barbour. 1941. Brown-spot needle disease of pines. Phytopathology 31:61-74.

3. Naemacyclus needle cast of pines

Butin, Von H. 1973. Morphological and taxonomic studies of *Naemacyclus niveus* and related species. European Journal of Forest Pathology 3:146-163.

Kistler, B. R., and W. Merrill. 1978. Etiology, symptomology, epidemiology, and control of Naemacyclus needlecast of Scotch pine. Phytopathology 68:267-271.

Kistler, B. R., and W. Merrill. 1978. *Naemacyclus minor*: High levels of infection in symptomless Scots pine. Phytopathology News 12(7):69.

Merrill, W., L. Zang, K. Bowen, and B. R. Kistler. 1979. *Naemacyclus minor* needlecast of *Pinus nigra* in Pennsylvania. Plant Disease Reporter 63:994.

4. Diplodia blight of pines

Brookhouser, L. W., and Glenn W. Peterson. 1971. Infection of Austrian, Scots and ponderosa pines by *Diplodia pinea*. Phytopathology 61:409-414.

Chou, C. K. S. 1976. A shoot dieback in *Pinus radiata* caused by *Diplodia pinea*. I. Symptoms, disease development, and isolation of pathogen. New Zealand Journal of Forestry Science 6:72-79.

Chou, C. K. S. 1976. A shoot dieback in *Pinus radiata* caused by *Diplodia pinea*. II. Inoculation studies. New Zealand Journal of Forestry Science 6:409-420.

Chou, C. K. S. 1977. Effect of tree age on *Diplodia pinea* infection of *Pinus radiata*. Plant Disease Reporter 61:101-103.

Chou, C. K. S. 1978. Penetration of young stems of *Pinus radiata* by *Diplodia pinea*. Physiological Plant Pathology 13:189-192.

Haddow, W. R., and F. S. Newman. 1942. A disease of the Scots pine *(Pinus sylvestris* L.*)* caused by *Diplodia pinea* Kickx associated with the pine spittlebug *(Aphrophora parallella* Say.*)*. Transactions of the Royal Canadian Institute 24(1):1-18.

Marks, G. C., and G. Minko. 1969. The pathogenicity of *Diplodia pinea* to *Pinus radiata* D. Don. Australian Journal of Botany 17:1-12.

Marks, G. C., and G. Minko. 1970. The resistance of *Pinus radiata* to infection by *Macrophoma pinea* (syn *D. pinea*). Australian Journal of Botany 18:55-65.

Peterson, Glenn W. 1974. Infection of shoots and cones of Austrian pine by *Diplodia pinea*. Proceedings of the American Phytopathological Society 1:110 [abstract].

Peterson, Glenn W. 1977. Infection, epidemiology, and control of Diplodia blight of Austrian, ponderosa, and Scots pines. Phytopathology 67:511-514.

Peterson, Glenn W. 1978. Effective and economical methods for controlling Diplodia tip blight. American Nurseryman 147(1):13, 66, 70, 72.

Peterson, Glenn W. 1981. Control of Diplodia and Dothistroma blights of pines in the urban environment. Journal Arboriculture 7:1-5.

Peterson, Glenn W. 1981. Diplodia blight of pine. USDA Forest Service, Forest Insect & Disease Leaflet 161, 7 p. Washington, D. C.

Peterson, Glenn W., and David S. Wysong. 1968. Diplodia tip blight of pines in the central Great Plains: Damage and control. Plant Disease Reporter 52:359-360.

Purnell, Helen M. 1957. Shoot blight of *Pinus radiata* Don caused by *Diplodia pinea* (Desm.) Kickx. Forests Commission of Victoria, Australia, Bulletin 5, 11 p.

Schweitzer, D. J., and W. A. Sinclair. 1976. Diplodia tip blight on Austrian pine controlled by benomyl. Plant Disease Reporter 60:269-270.

Slagg, Charles W., and Ernest Wright. 1943. Diplodia blight in coniferous seedbeds. Phytopathology 33:390-393.

Walla, James A., and Glenn W. Peterson. 1976. *Dothistroma pini* and *Diplodia pinea* not affected by surface wax of pine needles. Plant Disease Reporter 60:1042-1046.

Waterman, Alma M. 1943. *Diplodia pinea*, the cause of a disease of hard pines. Phytopathology 33:1018-1031.

5. Western gall rust of pines

Anderson, Gerald W., and David W. French. 1965. Differentiation of *Cronartium quercuum* and *Cronartium coleosporioides* on the basis of aeciospore germ tubes. Phytopathology 55:171-173.

Christenson, John A. 1968. A cytological comparison of germinating aeciospores in the *Cronartium coleosporioides* complex. Mycologia 60:1169-1170.

Hiratsuka, Y. 1969. *Endocronartium*, a new genus for autoecious pine stem rusts. Canadian Journal of Botany 47:1493-1495.

Kinloch, Bohun B., Jr. 1972. Genetic variation in resistance to *Cronartium* and *Peridermium* rusts in hard pines. p. 445-462. *In* Biology of rust resistance in forest trees. U. S. Department of Agriculture, Miscellaneous Publication 1221, 681 p. Washington D. C.

Kistler, B. R., and W. Merrill. 1978. Seasonal development and control of pine-pine gall rust. American Christmas Tree Journal 22:21-23.

Merrill, W., and B. R. Kistler. 1976. Phenology and control of *Endocronartium harknessii* in Pennsylvania. Phytopathology 66:1246-1248.

Peterson, Glenn W. 1973. Dispersal of aeciospores of *Peridermium harknessii* in central Nebraska. Phytopathology 63:170-172.

Peterson, Glenn W., and Richard S. Smith, Jr. 1975. Forest nursery diseases in the United States. U. S. Department of Agriculture, Agriculture Handbook 470, 125 p. Washington, D. C.

Peterson, Glenn W., and Ralph A. Read. 1977. Epidemiology of western gall rust in ponderosa pine provenance plantings in south central Nebraska. Proceedings of American Phytopathological Society 4:110 (abstract).

Peterson, Roger S. 1960. Western gall rust on hard pines. U. S. Department of Agriculture, Forest Service, Forest Pest Leaflet 50, 8 p. Washington, D. C.

Peterson, Roger S. 1967. The Peridermium species on pine stems. Bulletin Torrey Botanical Club 94:511-542.

Peterson, Roger S. 1971. Wave years of infection by western gall rust on pine. Plant Disease Reporter 55:163-167.

True, R. P. 1938. Gall development on *Pinus sylvestris* attacked by the Woodgate Peridermium and morphology of the parasite. Phytopathology 28:24-49.

Walla, J. A., and R. W. Stack. 1979. Western gall rust in North Dakota. Plant Disease Reporter 63:432-433.

6. Phomopsis blight of junipers

Hahn, Glenn Gardner. 1943. Taxonomy, distribution, and pathology of *Phomopsis occulta* and *P. juniperovora*. Mycologia 35:112-129.

Otta, J. D. 1974. Benomyl and thiophanate methyl control Phomopsis blight of eastern redcedar in a nursery. Plant Disease Reporter 58:476-477.

Otta, J. D. 1978. Alpha and beta spore occurrence in *Phomopsis juniperovora* pycnidia on *Juniperus virginiana* and in culture. Canadian Journal of Botany 56:727-728.

Peterson, Glenn W. 1967. Cedar blight. p. 55-56. *In* Important forest insects and diseases of mutual concern to Canada, the United States and Mexico. Canada Department of Forestry and Rural Development, Ottawa, Canada. 248 p.

Peterson, Glenn W. 1972. Chemical control of Phomopsis blight of junipers: a search for new methods. Tree Planters' Notes 23(3):3-4.

Peterson, Glenn W. 1973. Infection of *Juniperus virginiana* and *J. scopulorum* by *Phomopsis juniperovora*. Phytopathology 63:246-251.

Peterson, Glenn W., David Nuland, and John L. Weihing. 1960. Test of four fungicides for control of cedar blight. Plant Disease Reporter 44:744-746.

Peterson, Glenn W., D. R. Sumner, and C. Norman. 1965. Control of *Phomopsis* blight of eastern redcedar seedlings. Plant Disease Reporter 49:529-531.

Peterson, Glenn W., and C. S. Hodges, Jr. 1975. Phomopsis blight of junipers. U. S. Department of Agriculture, Forest Service, Forest Pest Leaflet 154, 5 p. Washington, D. C.

Peterson, Glenn W. 1975. Phomopsis blight of junipers. p. 76-79. *In* Forest nursery diseases in the United States. U. S. Department of Agriculture, Agriculture Handbook 470, 125 p. Washington, D. C.

Peterson, Glenn W., and J. D. Otta. 1979. Controlling Phomopsis blight of junipers. American Nurseryman 149(5):15, 75, 78, 80-82.

Peterson, Glenn W. 1963. Cedar blight. p. 105-106. *In* Internationally dangerous forest tree diseases. U. S. Department of Agriculture, Miscellaneous Publication 939, 122 p. Washington, D. C.

Peterson, Glenn W. 1965. Field survival and growth of Phomopsis-blighted and non-blighted eastern redcedar planting stock. Plant Disease Reporter 49:121-123.

Schoeneweiss, Donald F. 1969. Susceptibility of evergreen hosts to the juniper blight fungus, *Phomopsis juniperovora*, under epidemic conditions. Journal of American Society of Horticultural Science 94:609-611.

Stoessl, A. 1967. Altersolanol A, a novel tetrahydroanthraquinone. Chemical Communications No. 7, p. 307-308.

Wheeler, M. M., D. M. S. Wheeler, and G. W. Peterson. 1975. Anthraquinone pigments from the phytopathogen *Phomopsis juniperovora* Hahn. Phytochemistry 14:288-289.

7. Cercospora blight of junipers

Hodges, Charles S. 1962. Comparison of four similar fungi from *Juniperus* and related conifers. Mycologia 54:62-69.

Hodges, Charles S. 1975. Cercospora blight of juniper. p. 80-82. *In* Forest nursery diseases in the United States. U. S. Department of Agriculture, Agriculture Handbook 470, 125 p. Washington, D. C.

Ito, Kazuo, Takao Kobayashi, and Kozo Shibukawa. 1967. Etiological and pathological studies on the needle blight of *Cryptomeria japonica*. III. A comparison between *Cercospora cryptomeria* Shirai and *Cercospora sequoiae* Ellis at Everhart. Bulletin of the Government Forestry Experiment Station, Meguro, 204:74-90.

Kelman, Arthur, Charles S. Hodges, and H. R. Garris. 1960. Needle blight of redcedar, *Juniperus virginiana* L. Plant Disease Reporter 44:527-531.

Kilpatrick, R. A., and Howard W. Johnson. 1956. Sporulation of *Cercospora* species on carrot leaf decoction agar. Phytopathology 46:180-181.

Peterson, Glenn W. 1977. Control of juniper blight caused by *Cercospora sequoiae juniperi*. American Nurseryman 145(12):13, 50-51.

Peterson, Glenn W. 1977. Epidemiology and control of a blight of *Juniperus virginiana* caused by *Cercospora sequoiae* var. *juniperi*. Phytopathology 67:234-238.

Peterson, Glenn W., and David S. Wysong. 1968. *Cercospora* blight of junipers: Damage and control. Plant Disease Reporter 52:361-362.

8. Kabatina blight of junipers

Brener, W. D., E. C. Setliff, and R. L. Norgren. 1974. *Sclerophoma pythiophila* associated with a tip dieback of juniper in Wisconsin. Plant Disease Reporter 58:653-657.

Funk, A., and A. C. Molnar. 1972. *Kabatina thujae* on yellow cedar in British Columbia nurseries. Canadian Forest Service Bi-Monthly Research Notes 28:16-17.

Hoffman, G. M., and F. Fliege. 1967. *Kabatina juniperi* als Ursache eines Zweigsterbens an verschniedenen Juniperusarten. Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz 74:587-593.

Ostrofsky, Andrea, and Glenn W. Peterson. 1977. Occurrence of *Kabatina juniperi* on *Juniperus virginiana* in eastern Nebraska. Plant Disease Reporter 61:512-513.

Ostrofsky, Andrea, and Glenn W. Peterson. 1979. Infection of *Juniperus virginiana* by *Kabatina juniperi*. Phytopathology 69:1040-1041 (abstract).

Ostrofsky, Andrea, and Glenn W. Peterson. 1981. Etiological and cultural studies of *Kabatina juniperi*. Plant Disease 65:908-910.

Schneider, R., and J. A. von Arx. 1966. Zwei neue, als Erreger von Zweigsterben nachgewiesene Pilze: *Kabatina thujae* n. g., n. sp. und *K. juniperi* n. sp. Phytopathologische Zeitschrift 57:176-182.

9. Root lesion nematodes

Weihing, John L., Robert Inman, and Glenn W. Peterson. 1961. Response of ponderosa and Austrian pine to soil fumigants and seed treatments. Plant Disease Reporter 45:799-802.

Peterson, Glenn W. 1962. Root lesion nematode infestation and control in a plains forest tree nursery. U. S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station, Research Note 75, 2 p. Fort Collins, Colo.

Peterson, Glenn W. 1964. Heat treatment of nematode-infested eastern redcedar roots. Plant Disease Reporter 48:862.

Peterson, Glenn W. 1970. Response of ponderosa pine seedlings to soil fumigants. Plant Disease Reporter 54:572-575.

Peterson, Glenn W. 1981. Pine and juniper diseases in the Great Plains. USDA Forest Service General Technical Report RM-86, 47 p. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

The results of research in the Great Plains on five diseases of pines (Dothistroma needle blight, brown spot disease, Diplodia tip blight, Naemacyclus needle cast, western gall rust) and three diseases of junipers (Phomopsis blight, Cercospora blight, Kabatina branch tip dieback) are presented.

Keywords: **Pinus, Juniperus,** tree diseases, Great Plains

Peterson, Glenn W. 1981. Pine and juniper diseases in the Great Plains. USDA Forest Service General Technical Report RM-86, 47 p. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

The results of research in the Great Plains on five diseases of pines (Dothistroma needle blight, Brown spot disease, Diplodia tip blight, Naemacyclus needle cast, western gall rust) and three diseases of junipers (Phomopsis blight, Cercospora blight, Kabatina branch tip dieback) are presented.

Keywords: **Pinus, Juniperus,** tree diseases, Great Plains

Peterson, Glenn W. 1981. Pine and juniper diseases in the Great Plains. USDA Forest Service General Technical Report RM-86, 47 p. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

The results of research in the Great Plains on five diseases of pines (Dothistroma needle blight, Brown spot disease, Diplodia tip blight, Naemacyclus needle cast, western gall rust) and three diseases of junipers (Phomopsis blight, Cercospora blight, Kabatina branch tip dieback) are presented.

Keywords: **Pinus, Juniperus,** tree diseases, Great Plains

Peterson, Glenn W. 1981. Pine and juniper diseases in the Great Plains. USDA Forest Service General Technical Report RM-86, 47 p. Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colo.

The results of research in the Great Plains on five diseases of pines (Dothistroma needle blight, Brown spot disease, Diplodia tip blight, Naemacyclus needle cast, western gall rust) and three diseases of junipers (Phomopsis blight, Cercospora blight, Kabatina branch tip dieback) are presented.

Keywords: **Pinus, Juniperus,** tree diseases, Great Plains

Rocky Mountains

Southwest

Great Plains

U.S. Department of Agriculture
Forest Service

Rocky Mountain Forest and Range Experiment Station

The Rocky Mountain Station is one of eight regional experiment stations, plus the Forest Products Laboratory and the Washington Office Staff, that make up the Forest Service research organization.

RESEARCH FOCUS

Research programs at the Rocky Mountain Station are coordinated with area universities and with other institutions. Many studies are conducted on a cooperative basis to accelerate solutions to problems involving range, water, wildlife and fish habitat, human and community development, timber, recreation, protection, and multiresource evaluation.

RESEARCH LOCATIONS

Research Work Units of the Rocky Mountain Station are operated in cooperation with universities in the following cities:

Albuquerque, New Mexico
Bottineau, North Dakota
Flagstaff, Arizona
Fort Collins, Colorado*
Laramie, Wyoming
Lincoln, Nebraska
Lubbock, Texas
Rapid City, South Dakota
Tempe, Arizona

*Station Headquarters: 240 W. Prospect St., Fort Collins, CO 80526